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THE PRODUCTION OF AMMONIA BY BACTERIA.[1]

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OBJECT OF INVESTIGATION.

THE chemical mechanism by which urea is split into ammonia is an enzyme reaction, familiar to the biochemist in the estimation of the blood urea content. It has long been known that some bacteria possess the capacity to split urea. A certain amount of work on the subject has been done, but the field of investigation seems mainly to have covered organisms obtained from the soil, and little has been done on the bacterial inhabitants of man.

The chemical equation for the reaction is as follows:

$$(NH_2)_2CO + 2H_2O \rightarrow (NH_4)_2CO_3$$

Urea — Ammonium carbonate

This investigation was undertaken to determine, firstly, whether the production of ammonia from urea could be used as an auxiliary basis for the differentiation of bacteria, and, secondly, whether a solid medium incorporating urea and an indicator could be used to produce coloured colonies of urea-splitting organisms. This might prove of practical use, should it add an additional distinguishing property to any of the organisms of the dysentery or enteritis subgroups.

The obvious field for fruitful results is in infections of the urinary tract, since here the organisms are in an environment particularly favourable to their urea-splitting activity. The urological entities likely to yield positive results are: (i) persistently alkaline urine, (ii) acute diffuse fulminating hæmorrhagic cystitis, (iii) incrustations, (iv) urinary calculi. Stock cultures are not suitable for investigation, owing to deterioration of urease.

[1] Work done at the British Postgraduate Medical School, London, in 1939 and 1940, and completed in Melbourne in 1941.

HISTORICAL REVIEW OF THE PRODUCTION BY BACTERIA OF AMMONIA FROM UREA.

A small amount of research on the production of ammonia from urea has been done from time to time since the discovery by Pasteur,[1] in 1876, that ammoniacal decomposition of urine was of bacterial origin. Schnitzler[2] (1893) described the production by bacteria of ammonia and carbon dioxide from urea. The influence of sugars on the fermentation process was observed by Brodmier[3] (1895), who stated that glucose added to the medium prevented the liberation by *Bacillus proteus* of ammonia from urea. On the other hand, in 1916 Jacoby[4] observed that the addition of glucose to the urea solution accelerated the decomposition of urea by *Bacillus proteus.*

Ishikawa[5] (1928) also investigated the influence of carbohydrates on the bacterial decomposition of urea, and in 1917 Geilinger,[6] investigating 72 urea-splitting organisms obtained from soil and manure, found that four were able to decompose urea in the absence of oxygen. Jacoby[7] (1917) obtained a dried preparation of a urea-splitting bacillus, in which urease could be preserved for a long time and was active in the presence of toluol. He stated that leucin was necessary for the production of bacterial urease. In 1923 Takahata[8] found that the optimum pH of Sorensen's solution for the decomposition of urea by *Bacillus proteus* was 7·0, and that urease was extracted from the dried bacterial preparation of the neutral phosphate solution and its activity was favoured by glycocoll. Lehr,[9] in 1926, concluded that beryllium chloride in the phosphate buffer solution increased the urease activity of *Bacillus proteus.* Thompson isolated the *Corynebacterium thompsoni*, which is a strong urea-splitter

and occurred in epidemic form in urinary tract fistula. It was described by Shaw and Hill[10] in 1925.

Laidley[11] (1930), investigating infective phosphaturia, carried out routine examinations of all bacteria grown in a series of 200 consecutive cases. He developed a technique for detecting the presence of urea-splitting organisms and found that 52 of the series of 200 causative organisms in infective phosphaturia were able to split urea. Forty-two of these were Gram-negative bacilli, and the remaining organisms were cocci. He was unable to classify the cocci. The bacilli were more easily classified. Earlam[12] (1930), using Laidley's technique in a study of urea-splitting staphylococci in urinary tract infections, also included in this investigation 50 organisms from the intestinal tract. He found that 28% of these were urea-splitters. Both Laidley and Earlam classified the urea-splitting bacilli after Bergey.[13] The only urea-splitting bacilli which they identified by a generic name was the Proteus group.

Topley and Wilson[14] (1936) gave a list of soil bacteria which produce ammonia from urea. Throughout this publication the ability or otherwise (if known) of organisms to produce ammonia is usually stated in the list of their characteristics; but none of the fæcal flora is specified as producing ammonia.

Mitchell and Levine,[15] in 1938, in a study of nitrogen availability as an aid in differentiating the coli-aerogenes group of bacteria, chose nucleic acid and its degradation products—among them, urea. The work was spread over seven years, and the material used consisted of human and fowl fæces, frozen eggs and water from swimming pools. The significant findings were that 92% of intermediate coliforms and 100% of aerogenes split urea.

Thompson and Schulte[16] (1939), investigating 100 urinary cultures, found that Proteus (13 strains), diphtheroids (seven strains) and *Bacterium morgani* (two strains) split urea. Hills[17] (1940), in an extensive study of the deaminating ability of *Bacterium typhosum* and *Corynebacterium diphtheriæ*, found that results with urea were negative.

The present investigation was commenced by the study of a genus which contains some members known to have the property of splitting urea. A preliminary examination was made, correlating ammonia productivity with other properties. The genus *Staphylococcus* was selected.

Material.

The sources of the material were widespread. Staphylococci were isolated from specimens sent to the laboratory of a large London general hospital from patients suffering from conjunctivitis, otorrhœa, tonsillitis, mastitis, pyelitis, puerperal pyrexia, vaginitis, salpingitis, erysipelas, osteomyelitis, discharging wounds and pulmonary tuberculosis.

Correlation of Properties.

Two hundred and fifty strains of staphylococci were isolated, and an attempt was made to classify the organisms by correlating (i) sugar fermentation, especially mannite, (ii) pigment formation, (iii) hæmolysis, (iv) virulence, (v) ammonia productivity.

Methods.

Sugar Fermentation.

It was found that the presence or absence of fermentation of salicin, glucose, sucrose, lactose and maltose, with the production of acid but no gas, were properties largely common to the whole series, and therefore not available as a criterion for differentiation. Therefore, in the compilation of Table I fermentation of mannite, which was selective and had a definite association with other properties, is the only sugar reaction considered.

Pigment Formation.

Every organism was incubated for eighteen hours at 37° C. on (*a*) a nutrient agar slope for the determination of pigment formation and virulence, (*b*) a blood agar plate for hæmolysis. The majority of cultures were readily classified as *aureus*, *albus* and *citreus* strains. Subculture on Löffler's serum slope increased pigment formation in border-line *aureus* strains. There was a fourth type comprising about 10% of the total number of staphylococci under investigation. This is the creamy staphylococcus, which is apparently an intermediate variation between the *albus* and *aureus* strains, but more allied by properties to the former.

Hæmolysis.

Recent work has shown that staphylococci exhibit three types of hæmolysin. The α toxin, usual in human strains, hæmolyses both rabbit and sheep cells at 37° C. The β toxin, occurring in bovine strains, hæmolyses sheep cells only if incubation is followed by refrigeration (hot-cold lysis), and has no hæmolytic activity on rabbit cells, as shown by Bryce and Rountree[18] (1936). The $\alpha\beta$ toxin produces hæmolysis of rabbit cells at 37° C. and hot-cold lysis of sheep cells, as shown by Cowan[19] (1938).

Strains producing α-hæmolysis were found to predominate. Hæmolysin production occurred indiscriminately with other properties in staphylococci, and the findings were unsatisfactory for inclusion in Table I, which shows the association of properties.

Coagulase Production.

Coagulase production was considered the presumptive test for pathogenicity on account of its constancy and ease of demonstration. Cruikshank[20] (1937) stated that coagulase production appeared to be the simplest criterion for pathogenicity. The coagulating property does not disappear with age, nor with frequent subculture. Staphylocoagulase is relatively heat stable, resisting for half an hour temperatures up to 100° C. It was the considered opinion of Blair[21] (1939) that the coagulase reaction alone was sufficient *in vitro* indication of the pathogenic potentiality of staphylococci and that fermentation of mannite supplied a valuable confirmatory test.

A loopful of a suspension from a slope was inoculated in 1·0 millilitre of a one in 10 dilution of human plasma in broth or saline solution. The mixture was incubated at 37° C. A loose fibrinous clot appeared in from one and a half to three hours. Solution of the clot appeared in two or three days. This is the method described by Cruikshank[20] (1937).

It was found that coagulase production was inversely associated with ammonia productivity (see Table II), and that the time in which a coagulase-positive reaction resulted varied from half an hour to twenty-four hours with different strains of staphylococci.

Ammonia Productivity.

The method recommended by Thompson and Schulte[16] (1939) for the estimation of ammonia productivity was adopted. The composition of the medium was as follows: peptone 0·2 gramme, sodium chloride 0·5 gramme, distilled water 100 millilitres, urea 0·5 gramme. The medium was prepared by dissolving the peptone and sodium chloride in the required amount of water. To each litre were added 20 millilitres of a 0·2% alcoholic solution of thymol blue. The initial pH was adjusted to 6·8. The medium was tubed in amounts of 10 millilitres and sterilized. To the sterile tubes enough 10% urea solution was added to make the concentration of urea 0·5%. The solution was sterilized by filtration with a Seitz filter. All organisms were inoculated in 10 millilitres of urea peptone water (0·5%), in 10 millilitres of the same medium with the indicator omitted (for qualitative estimation of ammonia) and in 10 millilitres of plain peptone water (0·2%) as a control. Quantitative estimations of the ammonia produced in urea peptone water and in any control tubes giving a positive reaction have been made by removing an aliquot, treating it with Nessler's reagent and estimating it colorimetrically.

The urea-splitting enzyme has been found to be intracellular. Figure I shows the rate at which urea was split by eight strains of staphylococci. It will be seen that ninety-six hours' incubation suffices for practical purposes for the splitting of urea.

The gradual deterioration of urease on standing renders stock cultures useless for a study of ammonia productivity. A series of quantitative estimations of the urea-splitting property of six strains of staphylococci are shown in Figure II. From this it appears that *Staphylococcus aureus* loses the ability to split urea more rapidly than *Staphylococcus albus*. This is confirmed by the finding that the most common urea-splitter among staphylococci is the *albus* variety, and that, as the pigmentation deepens, the ability to produce ammonia decreases (see contingency tables, VIII to IX). All cultures show a considerable diminution of urease activity within two weeks of isolation. Revival of urease by passage through nutrient media was described by Hills[117] (1940).

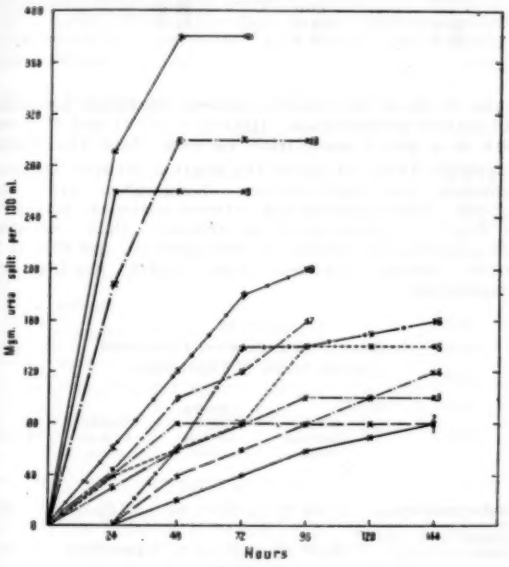

FIGURE I.

Showing the rate at which urea was split by eight strains of staphylococci. Three strains of *Proteus vulgaris* are included for comparison. 1, 2, 3, 4, 5, 6, 7, 8: *Staphylococcus albus*; 9, 10, 11: *Proteus vulgaris*.

Correlation of Properties.

To correlate the study of the properties under investigation Tables I and II have been compiled. The analysis comprises 250 strains of staphylococci in which observations have been made on each of the properties under consideration.

Table I shows the actual association of the properties in this sample (N) and also corresponding theoretical figures calculated on the assumption of complete independence, which serve as a comparison.

Table II gives in percentages the incidence of characters over a series of organisms, each such sample being constituted upon the basis of the presence of one of the above-mentioned properties.

By reference to Table I, the degree of association of properties evident in this series may be shown by the use of Pearson's index of association (Fisher, 1938).[cm] Contingency tables have been prepared (Tables III to IX).

Table III shows the relation between ammonia productivity and coagulase productivity for the 250 strains of staphylococci used. The number in the respective cells

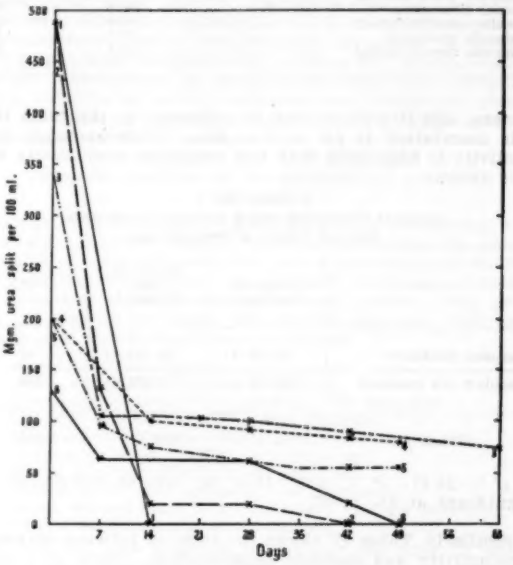

FIGURE II.

Showing the rate at which ability to split urea was lost by six cultures of staphylococci. 1, 2: *Staphylococcus aureus*; 3, 4, 5, 6: *Staphylococcus albus*.

indicates that, for example, 31 strains produced both ammonia and coagulase. The expected values are given in parentheses in each case. The value of χ^2 (Pearson's index of association) is calculated on the hypothesis that no association exists between ammonia productivity and coagulase productivity. This was found to be 24·47. The corresponding value of *P*, the probability that so large a value of χ^2 would be found purely by chance, is less than 0·01. There is therefore an association between the two

TABLE I.

Association of Properties within the Sample N.[1]

	Staphylococcus aureus.	Creamy *Staphylococcus albus.*	*Staphylococcus albus.*	*Staphylococcus citreus.*	Coagulase.		Mannite.		Ammonia.	
					Producing.	Non-producing.	Fermenting.	Non-fermenting.	Producing.	Non-producing.
Number of individuals possessing each attribute	109	23	117	1	96	154	138	112	134	116
Staphylococcus aureus (109 strains)	—	—	—	—	87	22	105	4	41	68
Creamy *Staphylococcus albus* (23 strains)	—	—	—	—	2	21	11	12	14	9
Staphylococcus albus (117 strains)	—	—	—	—	7	110	21	96	78	39
Staphylococcus citreus (1 strain)	—	—	—	—	0	1	1	0	1	0
Coagulase producing	(42)	(8·9)	(45·1)	(0·38)	—	—	95	1	31	63
Coagulase non-producing	(67·0)	(14·0)	(71·9)	(0·6)	—	—	43	111	103	53
Mannite fermenting	(60·0)	(12·7)	(64·4)	(0·55)	(53·0)	(85·0)	—	—	56	80
Mannite non-fermenting	(49·0)	(10·3)	(52·6)	(0·45)	(43·0)	(69·0)	—	—	78	36
Ammonia producing	(58·2)	(12·3)	(62·5)	(0·54)	(50·4)	(83·0)	(72·9)	(61·0)	—	—
Ammonia non-producing	(50·5)	(10·7)	(54·5)	(0·45)	(43·6)	(72·0)	(63·0)	(52·9)	—	—

[1] N: Total number in the series, 250. The figures not in parentheses are the actual observed figures; the figures in parentheses are those calculated for the case of independence of properties, given for purposes of reference.

TABLE II.

The Distribution of Characters in Populations Possessing the Attribute Denoted in the Column on the Left (Percentages).

—	Coagulase.		Mannite.		Ammonia.	
	Producing.	Non-producing.	Fermenting.	Non-fermenting.	Producing.	Non-producing.
Whole series of 250 organisms	38·0	62·0	55·0	45·0	54·0	46·0
Staphylococcus aureus series	35·0	9·0	42·0	1·8	16·4	27·0
Creamy *Staphylococcus albus* series	8·0	8·6	4·4	4·6	5·6	3·6
Staphylococcus albus series	2·2	44·0	8·2	38·4	32·0	15·6
Staphylococcus citreus series	0	0·4	0·4	0	0	0·4
Coagulase producing	38·0	—	34·4	0·4	12·4	22·4
Coagulase non-producing	—	62·0	13·2	44·4	39·0	23·4
Mannite fermenting	34·4	13·2	55·0	—	21·0	30·4
Mannite non-fermenting	0·4	44·4	—	45·0	29·6	14·4
Ammonia producing	12·4	39·0	21·0	29·6	54·0	—
Ammonia non-producing	22·4	23·3	30·4	14·4	—	46·2

factors, and it will be seen by reference to the table that this association is an inverse one. High ammonia productivity is associated with low coagulase productivity and *vice versa*.

TABLE III.

Ammonia Productivity versus Coagulase Productivity.

For 250 Strains of Staphylococci.

—	Ammonia Produced.	Ammonia Not Produced.	Totals.
Coagulase produced ..	31 (50·4)	63 (43·6)	94
Coagulase not produced	103 (83·6)	53 (72·4)	156
Totals	134	116	250

$\chi^2 = 24{\cdot}47$; $P < 0{\cdot}01$—that is, inverse association is significant at 1% level.

Similarly, Table IV shows the relation between ammonia productivity and mannite fermentation. Here $\chi^2 = 17{\cdot}43$ and $P < 0{\cdot}01$. There is therefore a significant inverse association between these two factors.

TABLE IV.

Ammonia Productivity versus Mannite Fermentation.

For 250 Strains of Staphylococci.

—	Ammonia Produced.	Ammonia Not Produced.	Totals.
Mannite fermented ..	56 (72·9)	80 (63·1)	136
Mannite not fermented ..	78 (61·1)	36 (52·9)	114
Totals	134	116	250

$\chi^2 = 17{\cdot}43$; $P < 0{\cdot}01$—that is, inverse association is significant at 1% level.

TABLE V.

Coagulase Production versus Mannite Fermentation.

For 250 Strains of Staphylococci.

—	Mannite Fermented.	Mannite Not Fermented.	Totals.
Coagulase produced ..	95 (53·0)	1 (43·0)	96
Coagulase not produced	43 (85·0)	111 (69·0)	154
Totals	138	112	250

$\chi^2 = 117{\cdot}97$; $P < 0{\cdot}01$—that is, direct association is significant at 1% level.

Table V shows the relation between coagulase production and mannite fermentation. Here $\chi^2 = 117{\cdot}97$ and $P < 0{\cdot}01$. There is a direct association between these two factors.

Likewise, Table VI shows the relation between coagulase production and pigmentation. Here $\chi^2 = 139{\cdot}49$ and $P < 0{\cdot}01$. Direct association between coagulase production and depth of pigmentation is evident. Only one strain of *Staphylococcus citreus* was encountered, and this is for obvious reasons omitted from tables dealing with pigmentation.

TABLE VI.

Coagulase Productivity versus Pigmentation.

For 249 Strains of Staphylococci.

—	*Staphylococcus aureus.*	Creamy *Staphylococcus albus.*	*Staphylococcus albus.*	Totals.
Coagulase produced..	87 (42·0)	2 (8·9)	7 (45·1)	96
Coagulase not produced	22 (67·0)	21 (14·1)	110 (71·9)	153
Totals	109	23	117	249

$\chi^2 = 139{\cdot}49$; $P < 0{\cdot}01$—that is, direct association exists between coagulase production and depth of pigmentation.

Similarly, Table VII shows the relation between mannite fermentation and depth of pigmentation. Here $\chi^2 = 140{\cdot}62$ and $P < 0{\cdot}01$. There is therefore direct association between these two factors.

TABLE VII.

Mannite Fermentation versus Pigmentation.

For 249 Strains of Staphylococci.

—	*Staphylococcus aureus.*	Creamy *Staphylococcus albus.*	*Staphylococcus albus.*	Totals.
Mannite fermented ..	105 (60·0)	11 (12·7)	21 (64·4)	137
Mannite not fermented	4 (49·0)	12 (10·3)	96 (52·6)	112
Totals	109	23	117	249

$\chi^2 = 140{\cdot}62$; $P < 0{\cdot}01$—that is, direct association exists between mannite fermentation and depth of pigmentation.

Lastly, Table VIII shows the relation between ammonia production and depth of pigmentation. Here $\chi^2 = 19{\cdot}66$ and $P < 0{\cdot}01$. It will be seen that the association between these two factors is an inverse one.

TABLE VIII.

Ammonia Productivity versus Pigmentation.

For 249 Strains of Staphylococci.

—	*Staphylococcus aureus.*	Creamy *Staphylococcus albus.*	*Staphylococcus albus.*	Totals.
Ammonia produced..	41 (58·2)	14 (12·3)	78 (62·5)	133
Ammonia not produced	68 (50·8)	9 (10·7)	39 (54·5)	116
Totals	109	23	117	249

$\chi^2 = 19{\cdot}66$; $P < 0{\cdot}01$—that is, an inverse association exists between ammonia production and depth of pigmentation.

These findings are summarized in Table IX.[1]

TABLE IX.

Properties.	χ^2	Association (in Each Case Significant at 1% Level).
Ammonia productivity and coagulase productivity	24·47	Inverse.
Ammonia productivity and mannite fermentation	17·43	Inverse.
Ammonia productivity and depth of pigmentation	19·66	Inverse.
Mannite fermentation and depth of pigmentation	140·62	Direct.
Coagulase productivity and depth of pigmentation	139·49	Direct.
Coagulase productivity and mannite fermentation	117·97	Direct.

THE FÆCAL FLORA.

The investigation was extended to the fæcal flora, in the hope that the presence of urease might provide an auxiliary basis for differentiation. The fæcal flora comprises those organisms usually found singly or severally in urinary tract infections.

Source of the Organisms.

Samples of fæces sent for examination for occult blood to a large public hospital provided the greater part of the material used (59%). These stools were from patients suffering from dyspepsia, gastric or duodenal ulcers and malignant disease. In most cases a meatless and iron-free diet was taken for three days before the stools were obtained. Normal stools provided a further 8%, and stools from patients with gastro-intestinal disturbances comprised 33%. Since it has already been shown that the ability to produce ammonia is gradually lost by cultures kept for some weeks, laboratory stock cultures could not be used; 168 organisms were investigated.

Methods.

A loopful of fæces was plated on MacConkey's bile-salt-lactose agar, and on blood agar. Routine incubation was aerobic, but occasionally blood agar plates were incubated anaerobically. Colonies were picked off and identified by the usual criteria.

Production of Ammonia.

The medium used was that of Thompson and Schulte[13a] (1939), identical with that described in the first part of this paper. A blue colour change indicated the production of ammonia. Quantitative estimations were not made.

The most significant findings are tabulated in Table X.

It is obvious that correlation between ammonia productivity and other properties is impossible in such a heterogeneous series of results. Liquefaction of gelatin, found by Laidley[11] to be a constant characteristic of strong urea-splitting bacilli, was not confirmed. Failure to liquefy gelatin on the part of all strains of ammonia-producing *Bacterium aerogenes* Type I and *Bacterium morgani* was noted.

To sum up: the incidence of pathogens in this investigation was disappointingly low, and little that is useful or new has been added to knowledge of the fæcal flora. The most commonly occurring organisms that produce ammonia from urea were *Bacterium aerogenes* Type I, *Bacterium morgani*, the Proteus group and *Staphylococcus albus*. Fæcal organisms that did not produce ammonia from urea were *Bacterium coli* and intermediate types, *Bacterium cloacæ*, paracolon bacilli, *Bacterium typhosum*, *Bacterium paratyphosum* B, *Clostridium welchii*, *Streptococcus fæcalis*, *Streptococcus fæcalis-lactis*, *Streptococcus pyogenes*, *Streptococcus viridans* and *Staphylococcus aureus*. Production of ammonia by *Bacterium aerogenes* Type I distinguishes this organism from *Bacterium cloacæ*, and may be used as a more rapid method of differentiation than liquefaction of gelatin.

Synthetic Medium for the Detection of Ammonia Productivity.

An attempt to elaborate a solid medium containing urea and an indicator which would differentiate by colour colonies of ammonia-producing and ammonia-non-producing bacteria has been made. A synthetic medium consisting of salts and a small amount of meat extract, with brom thymol blue as indicator, was finally selected. The composition of the medium was as follows:

Agar: 25·00 grammes.
Aqua destillata: 1,000·00 millilitres.
Potassium phosphate (K_2HPO_4): 1·00 gramme.
Calcium chloride ($CaCl_2$): 0·10 gramme.
Magnesium sulphate ($MgSO_4.7H_2O$): 0·10 gramme.
Sodium chloride (NaCl): 0·10 gramme.
Ferric chloride ($FeCl_3$): 0·01 gramme.
"Lab-Lemco" beef extract: 5·00 grammes.
Brom thymol blue: 10·00 millilitres of 1·5% solution.
Urea ($CO(NH_2)_2$): 20·00 grammes.

The medium was prepared by melting the agar in water and adding ingredients (excepting urea) in the order listed. After sterilization by steaming the urea was added. The pH of the mixture is 7·0 and no adjustment is required. Plates or slopes may be poured and left for some weeks before use. It is inadvisable to reheat flasks of stock medium more than once.

Results.

Plates of this synthetic medium are leaf green in colour. Inoculation with an ammonia-producing organism turns the medium deep Prussian blue. Only the full range of the brom thymol blue denotes a positive result. A large series of ammonia-producing and ammonia-non-producing organisms was tested. The colour change was produced in one hour with strong urea-splitting organisms, such as *Proteus vulgaris*, the spreading of which was inhibited. A strong smell of ammonia could be detected when the lid of the Petri dish was removed. The liberation of the gas, with consequent diffusion over the surface of the medium, results in the alkaline colour change of the dye.

Differentiation of colony coloration was evident. Thus ammonia-productivity was associated with grey-blue colonies, and ammonia-non-productivity with golden colony formation. The growth of most organisms is slightly inhibited and colonies may be small. Experiments proved that this was due to brom thymol blue and not to the urea or its breakdown products.

DISCUSSION.

The preceding observations partly confirm previous findings on urea-splitting bacteria, and partly differ from them. The investigation on staphylococci agrees with the work of Laidley[11] (1930) and Earlam[13a] (1930), in that it has been found impossible to classify staphylococci on the basis of their ability to produce ammonia from urea. The evidence of ammonia production was collected by these

[1] The discussion is placed at the end of the paper, where production of ammonia is dealt with as a whole in relation to all the organisms under consideration.

TABLE X.
Showing Properties of the Fæcal Flora.
(168 Organisms.)

Index Number.	Number of Strains.	Production of Ammonia.	Indol.	Methyl Red.	Voges-Proskauer.	Utilization of Citrate.	Liquefaction of Gelatin.	Motility.	Litmus Milk.	Lactose.	Glucose.	Maltose.	Mannite.	Heat Resistance.	Morphology.	Organism.
1–64	64	—	..	+	—	—	—	..	Acid.	A.G.[1]	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	*Bacterium coli.*
65	1	—	—	+	—	+	—	..	Acid.	A.G.	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	Bacterium Intermediate Type I.
66	1	—	+	+	—	+	—	..	Acid.	A.G.	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	Bacterium Intermediate Type II.
67–76	10	+	—	—	+	+	—	..	Acid.	A.G.	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	*Bacterium aerogenes* Type I.
77–80	4	—	—	—	+	+	+	..	Acid.	A.G.	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	*Bacterium cloacæ.*
81–110	30	—	+	+	—	—	—	..	Acid.	—	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	Paracolon bacillus.
111	1	—	—	..	..	+	—	+	Alkaline.	—	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	*Bacterium paratyphosum* B.
112–114	3	—	—	..	..	..	—	+	Unchanged.	—	A.	A.	A.	..	Gram - negative bacillus.	*Bacterium typhosum.*
115	1	—	—	+	—	+	—	+		—	A.	A.	A.	..	Gram - negative bacillus.	Unidentified N.L.F.
116	1	+	—	..	..	+	—	+	Acid.	—	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	Unidentified N.L.F.
117	1	—	—	..	..	..	—	+	Alkaline.	—	A.G.	A.G.	A.G.	..	Gram - negative bacillus.	Lesser known *Salmonella.*
118–126	9	+	—	+	—	+	+	..	Peptonized.	—	A.G.	—	—	..	Gram - negative bacillus.	*Proteus vulgaris.*
127–128	2	+	+	+	—	+	+	..	Peptonized.	—	A.G.	A.G.	—	..	Gram - negative bacillus.	Proteus X strain.
129–132	4	+	+	..	..	—	—	..	Alkaline.	—	A. (G.)	—	—	..	Gram - negative bacillus.	*Bacterium morgani.*
133	1	+	..	..	..	+	—	+	Acid.	A.[2]	A.	A.	A.	..	Gram - positive bacillus.	Diphtheroid.
134–148	15	—	..	..	..	..	..	..	Stormy clot.	A.G.	A.G.	A.G.	—	+ at 80°	Gram - positive bacillus.	*Clostridium welchii.*
149–157	9	—	..	..	..	..	..	..	Acid.	A.	..	..	A.	++	Gram - positive diplococci.	*Streptococcus fæcalis.*
158–160	3	—	..	..	..	..	..	..	Acid.	A.	..	..	—	—	Gram - positive cocci (chains).	*Streptococcus viridans.*
161	1	—	..	..	..	..	..	..	Acid.	A.	..	..	—	—	Gram - positive cocci (chains).	*Streptococcus fæcalis-lactis.*
162	1	—	..	..	..	..	..	..	Acid.	A.	..	..	—	—	Gram - positive cocci anaerobe, hæmolysin formed.	*Streptococcus pyogenes.*
163–164	2	—	..	..	..	..	..	..	Acid.	A.	A.	A.	A.	—	Gram - positive cocci.	*Staphylococcus aureus.*
165–168	4	+	..	..	..	..	..	..	Acid.	A.	A.	A.	—	—	Gram - positive cocci.	*Staphylococcus albus.*

[1] A.G. = Acid and gas. [2] A. = Acid.

two observers only from staphylococci occurring in the urinary tract, an environment particularly suited to their urea-splitting activity. The present investigation deals with staphylococci from lesions throughout the human body, and might reasonably be expected to provide greater variety. Yet no essential differences in characteristics of the ammonia-producing staphylococci have been found.

By a correlation of properties I have been able to demonstrate that ammonia productivity is inversely associated with pathogenicity in staphylococci. This correlation of properties has also shown pathogenicity to be directly associated with fermentation of mannite and depth of pigmentation. Ammonia productivity would therefore be expected to be, and is, inversely associated with these two properties.

These results refer to the splitting of urea by staphylococci. When the investigation was extended to the splitting of urea by the fæcal flora, results were disappointing, since the incidence of pathogens was low. No cases of dysentery occurred during the period of work, and the urease activity of many of the salmonella organisms and of the dysentery group in freshly isolated culture is still unstudied.

Positive findings of interest are the production of ammonia by all strains of *Bacterium morgani* encountered and the production of ammonia by *Bacterium aerogenes* Type I, an organism hitherto distinguishable from *Bacterium cloacæ* only by the fact that it does not liquefy gelatin. Ammonia productivity is a quicker method of differentiation, especially if the solid medium described in this paper is used. It is of interest to note that Laidley[(11)] stated that all strong urea-splitters liquefied gelatin, though feeble splitters sometimes failed to do so. I am unable to confirm this, for none of the ten strains of *Bacterium aerogenes* Type I nor the four strains of *Bacterium morgani* liquefied gelatin.

Each member of any particular type of the fæcal flora behaved similarly as regards urease activity; this finding contrasts with a great lack of homogeneous behaviour in the genus *Staphylococcus*, in which urease activity is inversely associated with pathogenicity. Thus 64 strains of *Bacterium coli*, 30 strains of paracolon bacilli, 15 strains of *Clostridium welchii*, nine strains of *Streptococcus fæcalis* and four strains of *Bacterium cloacæ* did not split urea, but 10 strains of *Bacterium aerogenes* Type I, four strains of *Bacterium morgani* and 11 strains of Proteus did.

Not only is ammonia a product of decomposition, but it may also be a source of nitrogen for growth. Fildes[(12)] (1940) pointed out that *Bacterium coli* can grow on ammonia, synthesizing protein, whereas *Bacterium typhosum* cannot. The medium used in this investigation is extremely simple, containing only a trace of peptone, sodium chloride and urea. All organisms grew well, except *Clostridium welchii*, which invariably grew poorly. Quantitative estimations showed that *Staphylococcus aureus* produced free ammonia equivalent to the whole of the urea present in the medium (Figure II).

As a means of differentiating urea-splitting bacteria in mixed culture, the advantages of a solid medium were apparent; coloured colonies of urea-splitting bacteria might be picked off from it. The synthetic medium described in this paper, containing traces of potassium, calcium, magnesium, sulphur, iron, sodium, phosphorus, meat extract and 2% of urea incorporated in agar with 0·015% of

brom thymol blue, resulted in differentiation of urea-splitting organisms. The medium turns deep Prussian blue, showing the full alkaline range of the indicator when ammonia is liberated by bacterial activity. Grey-blue colonies of urea-splitting organisms are easily distinguished from the golden colonies of non-splitters. The indicator is somewhat inhibitory and colonies are small. The presence of urea-splitting bacteria is never in doubt, however, the Prussian blue colour of the medium developing in a few hours with a strong urea-splitter. Easy to prepare and rapid in reaction, this medium is to be preferred to the urea peptone water hitherto used.

CONCLUSIONS.

1. In a series of 250 strains of staphylococci it has been found impossible to classify the organisms on the basis of their ability to produce ammonia from urea.

2. The ability to produce ammonia from urea is independent of pigment formation, production of coagulase (pathogenicity), and fermentation of mannite.

3. The production of ammonia is inversely associated with these three properties.

4. Depth of pigment formation, production of coagulase and fermentation of mannite are properties bearing direct association in staphylococci.

5. Fæcal bacteria which produce ammonia from urea are *Bacterium aerogenes* Type I, *Bacterium morgani*, the Proteus group and *Staphylococcus albus*.

6. *Bacterium aerogenes* Type I may be differentiated from *Bacterium cloacæ* on the basis of ammonia production.

7. Fæcal bacteria which do not produce ammonia from urea are *Bacterium coli* and intermediate Types I and II, *Bacterium cloacæ*, paracolon bacilli, *Bacterium typhosum*, *Bacterium paratyphosum* B, *Clostridium welchii*, *Streptococcus pyogenes*, *Streptococcus fæcalis*, *Streptococcus fæcalis-lactis* and *Streptococcus viridans*.

8. A solid synthetic medium containing urea and brom thymol blue indicator differentiates bacteria which produce ammonia from those which do not.

9. This medium is easier to prepare, and results in more rapid detection of urea-splitting activity, than the urea peptone water hitherto used.

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THE TREATMENT OF BURNS.[1]

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WHILE there are more than a few surgeons who complacently regard the results of their treatment of most diseases as satisfactory, probably none would dare to affirm that the immediate or late results of their treatment of other than the mildest of burns were such that further improvement was unlikely. The increased interest in the subject of burns now being displayed in England, as exemplified by the discussion at the Royal Society of Medicine, London, on November 6, 1940, and the improved results which have followed and which will in the future undoubtedly follow this interest, are a few of the minor benefits which we may hopefully expect to derive from the present war.

Shock, Toxæmia and Sepsis.

The three well known factors responsible for the fatalities accompanying burns are shock, toxæmia and sepsis. The last-mentioned is often quite obviously due to hæmolytic streptococci; but as to the causes of the other two, shock and toxæmia, while there are many theories, no agreement has yet been reached. The application of coagulants to burnt surfaces was certainly a notable advance in the treatment of burns. This method was introduced to lessen the dangers of the triad shock, toxæmia and sepsis, and except in certain situations and types of burns in which its use is ineffectual or dangerous, this type of treatment relieves pain, lessens the exudation of fluid, decreases the autolysis of the tissues and produces conditions inimical to the growth of bacteria. Morphine and warmth are the essentials in the treatment of primary shock, whereas in secondary shock, since the most potent factor in its causation is the great loss of plasma from

[1] Read at a Library Seminar held at Prince Henry Hospital on December 8, 1941.

the burnt surfaces and into the tissues (the latter being the more important of the two), intravenous administration of plasma or serum is indicated in order to raise the plasma osmotic pressure, to maintain the normal distribution of fluid between the blood and the tissues and to reduce the hæmoconcentration of the blood.

Wilson[1] found a lowering of the serum sodium level during secondary shock following burns, and treatment with desoxycorticosterone acetate tended to elevate the serum sodium level, with coincident improvement of the patient's clinical condition. Wilson therefore recommended the use of this drug in addition to plasma transfusions in the treatment of severe shock. The use of the "B.L.B." mask to attain a high alveolar oxygen tension has helped to lessen the mortality rate from shock and to reduce the incidence of fat embolism (quite a frequent occurrence in fatal cases of burns). This high alveolar oxygen tension is also of importance if in addition to the burn there is an associated "blast lung". Glucose infusions, even in large quantities, scarcely affect the hæmoconcentration and are indicated only to protect the liver during severe toxæmia. Even then, only small quantities should be used. Aldrich suggested that the acute toxæmia of burns was due to infection by streptococci; but while infection undoubtedly frequently plays a part in the production of the toxæmia of burns in the majority of cases, there are some cases in which the toxæmia develops in the absence of infection. According to Wilson,[1] the hypothesis of absorption of autolytic products from the burnt area is not proven. The most serious objection to it is that completely aseptic autolysis does not produce potent toxins. Although the toxæmia of burns, when fatal, is always accompanied by bacterial infection, yet liver lesions such as are found in these fatal cases of burns are never produced by common bacterial infections, even of the most severe type. It may be, however, that infection and absorption together result in the production of the toxæmia. The coagulation of the surface of the burn definitely inhibits the growth of the organisms and diminishes the absorption from the burnt area, and should therefore be doubly effective in lessening toxæmia. If a serious attempt is to be made to reduce the incidence of sepsis in burns, the burnt area should be treated by the same careful aseptic routine method, both for the first and for later dressings, as an ordinary wound would be.

Treatment.

Professor McIndoe[2] has pointed out that coagulation therapy has often been used in cases in which it is unwarranted or in which it is definitely contraindicated. The production of a coagulum over a burnt area has been likened by Hudson to primary suture of a surgical wound without drainage, and therefore such treatment should not be used indiscriminately. When the burn is of only the first or the second degree, the method of coagulation gives excellent results provided that sepsis can be excluded. In cases in which the loss of skin is of the third degree (that is, full thickness), it is impossible to coagulate the whole burnt area and sepsis inevitably occurs beneath the superficial coagulated layer. Thus, instead of being extinguished, the septic process may be considerably enhanced, with the result that months may elapse before the slough separates. Even then, healing is painful and accompanied by dense scarring, anæmia and general debility of the patient. Localized third degree burns are probably best treated, in the light of present knowledge, by immediate excision and grafting. If they are too extensive for excision, the best treatment then available is either the recently described envelope method or the use of saline baths and saline dressings, followed in each case by grafting. In this war most of the burns have been on the exposed parts of the body (the hands and the face)—areas usually unsuitable for coagulation therapy. Because the tannic acid jellies when used at the first aid posts are often applied hurriedly and inadequately over unclean burnt surfaces without any consideration of the depth of the burn, McIndoe[2] recommends a simpler application, such as gentian violet jelly, with which it is usually possible later to determine the depth of the burn. An Emergency Medical Services memorandum issued by the Medical Research Council of the Privy Council suggests that after being cleansed and dried the burnt area should be dusted with sulphanilamide before the coagulant is applied. After the use of tannic acid preparations as the final dressing in special situations, such as on the hands, movement of the fingers may be limited for months and pressure effects may develop. As McIndoe[2] pointed out, the œdema developing beneath the coagulum as a result of the burn immobilizes the tendon sheaths very early and is a definite factor in causing fixation of the joints. Sepsis, when present, further increases the œdema. The pressure effect is also intensified by the fixed encrusted tan which does not yield with the increase in size of the part. Embarrassment of the circulation of the fingers leads to crippling deformities, such as spindle fingers, partial necrosis or even partial loss of digits. A common complication is pressure necrosis of the tissues overlying the proximal interphalangeal joints with perforation into the joints. The final result is then a fixed, clawed, useless hand. Such a chain of events may happen with a burn of the second degree if it is converted into a third degree burn by necrosis. On the eyelids, if the burn is of the third degree, and unless grafting is carried out early, sepsis is inevitable, with rapidly developing ectropion and exposure of the globe, corneal ulceration and perforation beneath the coagulum.

For the first aid treatment of burns Wakeley[3] favours gential violet jelly with merthiolate (1 in 5,000). This may be dispensed in a collapsible tube and is therefore unlikely to be spoilt by water, oil, grease *et cetera*. Being an efficient antiseptic, it has the advantage that it may be temporarily applied to the burnt area without any cleansing, as may be necessary during the stress of battle. It is also painless, soothing and effective. As a coagulating agent the aqueous solution of tannic acid has been almost completely replaced by such combinations as gentian violet (1%), silver nitrate (10%) and tannic acid (15%). Triple dye, which is a solution containing 1% (or 2%) of gentian violet, 1% of brilliant green and 0·1% of neutral acriflavine, is sprayed or painted on the injured surface after cleansing with saline solution and excision of all blistered and dead epidermis. Ether soap should be used for cleansing the burnt area only when an oily or greasy application has been used as the first aid dressing. At all times it is important that the body of the burnt patient is not unduly exposed during the cleansing and dressing of his burns. This especially applies to the patient on whom it is necessary to use ether soap during the cleansing of the burn. It is said that two or three applications of triple dye will result in a nice, thin, supple tan; but it is sometimes found that several applications are necessary before a suitable tan is acquired. If, as sometimes happens, sepsis appears at the edge of the triple dye tan, cleansing and retanning under anæsthesia are sometimes recommended; but the application of saline dressings over paraffin-impregnated net and grafting later are usually to be preferred, for the burn by the time sepsis develops has become of the third degree. Many authors who advocate the use of triple dye seem to have overlooked the occurrence of infection beneath the tan; but there is no doubt that in insufficiently cleansed areas it happens frequently. For the late after-treatment of burns, Wakeley[3] suggests that lanolin be rubbed into the healed surface each day for at least six weeks. This massage is said to result in a skin which is more supple and more vascular. In THE MEDICAL JOURNAL OF AUSTRALIA of October 25, 1941, de Vidas and McEachern[4] reported the use of a tannic acid jelly containing 7·5% of sulphanilamide. They found that the absorption of the sulphanilamide continued until the coagulum of the tannic acid formed, after which absorption ceased. These results, when translated to the probable absorption of autolysed tissue products, would confirm the advisability of producing a tan whenever possible. Robson and Wallace[5] have recently described the good results obtained with a glycerin-sulphonamide paste containing "Albucid", glycerin, kaolin and cod liver oil for local application to burns of the hands and face. On theoretical grounds its use should be attended by

good results, and further trial of this paste is indicated. In the treatment of burns of the hands and parts subjected to pressure, good results have followed the use of a single application of triple dye after the burnt area has been cleansed with saline solution, then dried and coated with a layer of flavine emulsion (Australian Pharmaceutical Formulary) about an eighth of an inch thick, the entire part being covered with a bandage which is left undisturbed for seven days. On removal of the dressing after this time, if the wound is not healed the dressing is repeated.

From a consideration of the factors essential in the healing of wounds in general and of burns in particular, Bunyan[6] has evolved a method of treatment of burns which consists of intermittent irrigation with weak solutions of electrolytic hypochlorites. The advantages of these solutions are: (i) their selective action dissolves the dead tissue and stimulates healing tissues; (ii) the breakdown products are salt and oxygen; (iii) the bactericidal powers are great in non-irritant concentrations; (iv) stock solutions are stable and of constant strength; (v) pain is relieved and desensitization is obtained. The method of treatment described by Bunyan[6] consists of hosing down the burnt area with a 5% solution of electrolytic hypochlorites to remove all surface contamination, much of the charred tissue and all the exudates, and to kill most of the bacteria present. A special envelope (Bunyan-Stannard) is then applied to the part for purposes of irrigation. These envelopes are made of fine silk coated with synthetic resins; they have an inlet and an outlet, and the open end of the envelope may be sealed around the part by adhesive silk, strapping and rubber or pneumatic bands. Bunyan[6] has used various sized envelopes up to those enclosing the body and limbs. Irrigation with 2·5% hypochlorite solution is carried out for twenty minutes three times per day until the infection is overcome, and then twice a day. When necessary, the part may be dried by blowing warm air or oxygen through the envelope. The envelope is left in place until epithelialization is complete. One of the advantages of the method is that the silk of the envelopes is transparent and the process of healing can be followed throughout its course. This method of treatment is also applicable to cases in which extensive burns coexist with compound fractures or multiple wounds. Since his original paper Bunyan[7] has again discussed this method, and up to that time he had satisfactorily treated 200 patients.

In third degree burns the method of soaking off sloughs by saline packs or by repeated or continuous saline baths produces a clean, granulating surface much more quickly than does the coagulation method. Saline baths for the whole body are useful for extensive mixed second and third degree burns. In the Royal Navy it has been noticed that those men suffering from burns who have been immersed in the sea suffer less pain and shock than would have been expected. The saline bath apparently exerts its beneficial effects whether intended or enforced.

The Sulphonamides.

The question of the prophylactic use of the sulphonamides in the treatment of burns has recently received attention. Although this treatment may have little effect on the local septic process, it has been recommended as a protection against the dangers of septicæmia and pyæmia. Bunyan[6] is of the opinion that chemotherapy is usually unnecessary when the envelope method is used, because of the disinfection and the actual prevention of toxæmia and secondary infection by this method. Wakeley[3] also thinks that the sulphonamides are rarely necessary in the treatment of burns, and points out that any vomiting induced by the drugs would further impair the patient's fluid balance. Colebrook[8] differs from these surgeons, for he thinks that there is great benefit to be derived from the local application of the sulphonamides, either as a powder or in some form of paste. In my limited experience it has been found that the local application of sulphanilamide to infected second degree burns was of use only in those cases in which the formation of pus or the exudation of fluid was limited in amount before the drug was applied. If the pus formation or the exudation of fluid was large in amount, eusol or saline pads gave better results than sulphanilamide powder.

Burns Due to Chemicals.

Special treatment is required for burns due to chemicals. For burns due to phosphorus one application of a 2·5% aqueous solution of copper sulphate followed by drying of the burnt area and then tanning with triple dye is recommended. In the absence of such treatment these burns are apt to become deeper as the phosphorus continues its thermal action on the tissues.

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NERVOUSNESS: A NEGLIGIBLE AND NOT PENSIONABLE DISABILITY.

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OF late years the psychologist has endeavoured to reduce the instinctive phenomena to more manageable proportions. This is a welcome phase of the scientific streamlining which has occurred during the last few years. Even at the risk of an accusation of taking a retrograde step by discussing another instinct, one for "tagging" is suggested for criticism and possibly adoption. The word describes man's instinctive fear of being confronted with something which cannot be placed in a neat little pigeon hole. Such an inherent tendency, whilst admirable in many scientific pursuits, has far-reaching and often very unfortunate repercussions. A man who is infected with even a small degree of liberal principles is tagged as a socialist. On the other hand, if he believes in the efficacy of elementary discipline, he is a Fascist. That this instinct is deeply rooted in racial consciousness is shown by such expressions as "give a dog an ill name and hang him", "once a thief, always a thief". These notes concern an important medical angle of tagging.

When is a Neurosis not a Neurosis?

The caption "when is a neurosis not a neurosis" is not an attempted witticism, but rather the simple expression of a common problem. The far-sighted constantly ask us to consider the very early signs and symptoms of disease. They point out that most text-book descriptions are those of end results, rather than processes. To use a homely metaphor, we frequently broach a used and empty keg.

To revert to the neurosis and psychoneurosis, beyond somewhat vague references to such entities as psychopathic personality, anxiety trends *et cetera*, our text-books of psychiatry are singularly reticent about the early stages, doubtless for the reason that they differ so little from normality. There is no definite borderline between the neurosis and health, since a stage is soon reached

wherein it is quite impossible to say whether or not a neurosis exists.

From one aspect it is distinctly unfortunate that text-books and learned papers are largely written by specialists, who encounter their patients in the disease "blossoming" rather than the disease "planting" stages. The condition antedating the neurosis is treated by general practitioners. It includes such manifestations as "being out of sorts", "feeling on edge", "a little jittery", "feeling worried", "head feeling numb", "shaky", "sleepless", "off food", "strung up" *et cetera*.

The diagnosis in such cases depends on the turn of circumstances. If the patient has the fortune to see an experienced general practitioner, nomenclature is evaded. His "you are just a little nervous and run down, you need a fortnight's holiday", is really an apt description. In the vast majority of cases the therapeutic test confirms the existence of a benign syndrome having a high recovery rate. On the other hand, if the patient's means are such as to permit a consultation at the hands of a more high-brow practitioner or a specialist, he will almost inevitably be written up in the case cards as having a neurosis or possibly even a psychoneurosis. The treatment may be identical.

At first sight the differences in diagnostic nomenclature may be regarded as those between Tweedle-dum and Tweedle-dee. The homely general practitioner merely treads with diagnostic ignorance in the wake of the wary consultant angel. The consultants play safe lest they might make an occasional mistake. Since the symptoms are those of a neurosis, they believe that it is legitimate to regard these conditions as minor examples of the classical syndrome. There is, however, a further possibility: the general practitioner may be entirely right in his attitude. The symptom complex may belong to a syndrome which is neither a neurosis nor a psychoneurosis. The underlying pathological condition may be merely a normal fatigue state or a conditioned anxiety, leading to increased awareness of self.

In my opinion, this view of the existence of a benign syndrome, which is not an anxiety neurosis, nor neurasthenia, nor a psychoneurosis, is correct. I make the suggestion that it be called by a name hallowed by long usage, that of "nervousness". For the suggestion to use this name I am indebted to Dr. W. H. Steel, who points out that its colloquialism leads to simplicity in explaining symptoms to the patient.

Some Examples of the "Nervousness" Syndrome.

M. Barnard Eldershaw, in the book "Phillip of Australia", describes the sad lot of the convicts on board the transport *Alexander*, almost ready to depart from England. They make the diagnosis of "mass hysteria". The prompt recovery under simple treatment suggests the existence of the "nervous syndrome". The following dialogue is dated 1787.

The first medical Gentleman: "I am very glad you are arrived, sir, for your people have got a malignant disease among them of a most dangerous kind, and it will be necessary for their preservation to get them immediately relanded."

Mr. Balmain: "Sir, you will not find things by any means as bad as this gentleman represents them to be; they are made much worse by him than they really are. Unlike a person wishing to administer comfort to those who are afflicted, either in body or in mind, he had publicly declared before the poor creatures that are ill, that they must inevitably fall a sacrifice to the malignant disorder with which they are afflicted—*the malignancy of which appears to me to exist only in his own imagination*. I did not, however, think proper to contradict the gentleman, supposing from the consequence he assumed and the ease with which he had given his opinion, or more properly his directions, that he was some person appointed by the Secretary of State for you until your arrival. When you go among the people, you will be better able to judge of the propriety of what I have said."

He prescribed fresh air, more fresh food, and a little wine for the more sick members. The epidemic rapidly subsided.

1917: A personal case history has the merit of a life-time intimacy. For this reason the best descriptions of symptoms are often those of medical men. A visit to the Dardanelles in 1915 as reinforcement for the Naval Division was ended by an attack of jaundice and dysentery. After a stay in hospital and some leave, the writer gravitated to a mine-sweeper. Later he noted many of the symptoms of the nervousness syndrome. They were never treated, but completely disappeared under a routine of work, change of scene and interest. They have never returned.

1941: The foregoing references have been introduced to show that the nervousness syndrome is not a modern innovation in a medical sense. It is time-honoured, and so ubiquitous that all medical men have encountered many cases, and a large number will have had the doubtful pleasure of personal experience. There is, however, a real difference, due to our attitude to disease based on the tagging instinct as mentioned in my opening remarks. The complex remains the same, but the descriptive tag has altered. This is a natural process; our grandfathers, when they wished to leave a situation hurriedly, termed the process "skedaddling". We "vamose", whereas our children "scram". Tomorrow they may "bale out".

Years ago colitis was a fashionable disease favoured by rich ladies. Today it has a very different significance. Prior to the last great war, neurasthenia was a mere nervous breakdown or prostration. Since that date more than two decades of military and civil pensioning aided by widespread and organized publicity have raised it into a new category. With anxiety neurosis, hypochondriasis and sundry other ailments, it forms a group of illnesses so serious as to warrant a lifetime of monetary compensation in the form of a pension. The old tag of neurasthenia has a new lease of life. But in its new setting it is dangerous. Suggestion, persuasion, imitation, the three psychological forces which batter our minds into acquiescence, have changed beyond recognition the mass ideology towards pensions. No longer is a pension a kindly aid for adverse circumstances to be accepted with gratitude; rather it is a right to be obtained if humanly possible. Moreover, any disease which is tagged with the pension label should logically entitle a man to obtain it. Were the process merely on the conscious level, counter-arguments might prevail; but unfortunately the sub-conscious steps in and creates symptoms to please the pension-minded. There is a real danger that this may be on such a thorough scale as to create an increasing disability which causes endless suffering to the patient. He must be guarded against the suggestibility of his own unconscious. The only way to do this is to make certain that our diagnostic criteria are correct and that we do not put a pension tag on to a non-pensionable entity.

Reports of Cases.

The following histories of returned soldiers of the present war are illustrative.

CASE I.—The patient has headache, jumpiness, depression, inability to concentrate. The symptoms have been accentuated through an accident on service and by an experience in a "blitz". The symptoms are those of "nervousness", and it is significant that some of them were present prior to enlistment on account of "domestic worry".

The official diagnosis was that of "anxiety state". It is my opinion that this descriptive term is psychologically unsound. This man has a background of "nervousness". He has had to work with it before, he will work again with equal facility, unless he is told that he has a pensionable disability. In the latter case, by suggestion a true neurosis, psychoneurosis or psychosis may be manufactured. The official label should run: "Nervousness constitutional. Disability negligible."

CASE II.—The symptoms are "tightness in the legs", "pins and needles in the knees", headaches, "twitching in the eye", "jumpiness", shakiness at times. The patient had had an exhausting period on service, and then had the ill luck to be in close proximity to an explosion. Symptoms commenced shortly afterwards.

When seen by me, the patient admitted that his condition had considerably improved, but said that he was still somewhat "jumpy". His "nervousness syndrome" is a normal result of his psychological traumata. The

tendency to recovery is certain unless the scales are tipped through an error in diagnosis. The annotation on his record card should run somewhat as follows: "This man has symptoms of a nervous type, but in my opinion they do not amount to an anxiety neurosis, nor can they be assessed as more than a negligible disability. They will disappear if he is found work in the near future."

The following type of case is beginning to find general recognition. When soldiers are transferred from the battle area on account of nervous disorders, there is apt to be a preliminary period of profound anxiety. The symptoms in this case are almost identical with those in Case II. The patient is being returned to Australia; after a few days at sea there is a remarkable improvement. Our patient literally "sparks up". He loses his "jitters". He becomes normal. He returns to a happy and interested life. Then as the ship nears the port of disembarkation, there is a noticeable change. Symptoms of "nervousness" reappear and accompany him to hospital.

These cases are valuable evidence as to the validity of my thesis. Are we to suppose that a serious morbid pathological condition is cured by a few days at sea, and that it reappears at sight of land? Rather is it that the nervousness normally disappears with rest and security, and reappears when the patient returns home and has unconscious anxiety as to saving his face. A healthy, happy man would savour of the coward deserting his "cobbers". Actually, he has been repatriated for nervousness.

It is inconceivable that morbid anxiety can be cured so quickly. Norman nervousness satisfactorily explains the riddle. The patient's "ups and downs" are but part of a normal process. No harm will be done unless he is unlucky enough to be treated as suffering from a grave nervous disorder.

Conclusion.

In conclusion, I must stress the fact that the foregoing remarks are made in the interest of the patient's health, and are not dictated by any need for national economy. Widespread recognition of "nervousness" as a normal, non-pathological accompaniment of psychic trauma, creating only negligible disabilities which time will dispel, will be of practical use to our patients. Treatment waits on diagnosis. Psychological treatment is largely a matter of explaining to the sufferer the nature of his symptoms. The moment he understands that they are normal reactions, and not morbid reactions, there is born, not merely the hope of recovery, but equally important, freedom from that major insecurity, the ever-present fear of losing a pension privilege.

Summary.

1. Attention is drawn to the necessity of avoiding the use of names or tags which provide an honourable excuse for unconscious self-deception.

2. "Nervousness" should be recognized as a normal, non-pathological sequela of psychic traumata, creating only negligible disabilities which time and occupation will eradicate.

3. It is suggested that every medical practitioner who deals with persons likely to be pensionable should make "nervousness" the first diagnostic entity to be considered. Only after this avenue has been fully explored is there need to consider those morbid phenomena which, in the past, have received a too ready recognition.

THE DETERMINATION OF THE THIAMIN (VITAMIN B_1) CONTENT OF THE DIET OF SERVICE PERSONNEL IN TRAINING.

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THE thiamin (vitamin B_1 or aneurin) content of a diet is usually determined theoretically. This calculation involves the use of "preferred" values for the thiamin content of the various foodstuffs. But tables such as those compiled by Fixsen and Roscoe[1] show that a foodstuff has no fixed vitamin content, but varies from sample to sample. Another difficulty in the evaluation of Australian diets is the necessity of using figures obtained for overseas products, which may differ considerably from local foods. For example, Slater and Rial[2] have shown that Australian wheat and white flour contain considerably more thiamin than foreign samples. The destruction of thiamin during cooking introduces a further error, as it is difficult to assess accurately the amount of the vitamin lost during the process.

Consequently, any determination on paper of the thiamin content of a diet gives only the theoretical average content. As pointed out by Clements, Slater and Rial,[3] such a determination is quite valid for the comparison of different diets, but is inaccurate when the absolute value of the thiamin content of a particular diet is required. Other work in this laboratory required such a determination.

In this study the food analysed was taken from that prepared for service personnel in training. Complete daily servings were analysed for thiamin by the thiochrome method. Williams and Spies[4] have shown that the thiamin:non-fat Calories ratio gives a reliable index of the status of a diet with respect to thiamin. Accordingly the diet was also analysed for fat, protein, ash and carbohydrate (the latter by difference).

Experimental Observations.

Collection of Samples.

The daily serving was the same as selected from the menu by the same person throughout. One day's food included the midday and evening meals of one day and breakfast of the next. The food was collected on four days in each week, the first commencing on Sunday at midday and the last ending on Thursday morning. The period covered was from May 6 till June 5. With the exception of the choice between rolled oats and cereals at breakfast, individual preferences would not have a marked influence on the amount of thiamin consumed. The person responsible for the collection of the food samples ate neither cereals nor rolled oats for breakfast. This point is further discussed below.

Beverages such as tea, coffee *et cetera* were not collected, but the sugar used in them was added to the bulked food and the milk collected in a separate container. Butter was also collected separately (according to the procedure of Wardlaw and White[5]), and the contribution of the milk and butter was calculated from the known composition of these foods.

The bulked food was collected in a tin container and arrived at the laboratory two hours after breakfast. It was immediately weighed and inedible material such as bones was removed. The food was then minced thoroughly. Samples of 40 grammes were taken for the thiamin estimations and the remainder was weighed and dried in a water oven for forty-eight hours at 70° C. The dried material was weighed, ground and stored in an air-tight container.

Method of Estimation of Thiamin.

Before the thiochrome method can be applied to extracts of natural products, free thiamin must be liberated from the phosphorylated (cocarboxylase) and protein-bound forms. Houston and Kon[6] have shown that incubation with takadiastase or with pepsin followed by takadiastase will bring this about.

Two methods of extracting the free thiamin from the minced foodstuff were compared, method *A* and method *B*. Method *A* was carried out as follows:

To 40 grammes of minced food were added 120 cubic centimetres of sodium acetate and acetic acid buffer solution, of pH 3·8, and the whole was heated at 70° C. on a water bath for fifteen minutes. After cooling, 0·5 gramme of takadiastase was added and the mixture was incubated at 37° C. for sixteen hours.

Method *B* was as follows:

Amounts of 40 grammes of minced food were heated with 120 cubic centimetres of 0·1 N hydrocholoric acid at 70° C. for fifteen minutes. After cooling 0·1 gramme of pepsin was

added and the whole was incubated at 37° C. for six hours. Sodium hydroxide was added to bring the pH to 3·8 (tested with bromphenol blue) and 0·5 gramme of takadiastase was added. The incubation was continued for a further sixteen hours. This method of extraction is essentially the same as that used by Pyke.[7]

After incubation the volume was made up to 20 cubic centimetres with distilled water. A portion was centrifugated and 5·0 cubic centimetres of the centrifugate were pipetted into a centrifuge tube of 15 cubic centimetres' capacity. This was shaken with five cubic centimetres of isobutanol and again centrifugated. Thiamin was determined on two cubic centimetres of the water layer by the visual method of Wang and Harris.[3] A constant amount of potassium ferricyanide (0·2 cubic centimetre of 2% solution) was used for the oxidation, the separate control test recommended by Wang and Harris being omitted (see Slater[9]). The standard thiochrome solution was prepared from two cubic centimetres of thiamin solution (one microgramme per cubic centimetre), oxidized by 0·05 cubic centimetre of 2% potassium ferricyanide solution.

Repeated analyses on several batches of 40 gramme samples of the minced food showed that 40 grammes were sufficient to give representative samples. Results are accurate to within about 10%. Booth[10] criticises the method of Pyke because the volume occupied by undissolved material after incubation introduces an uncertainty in the volume of the aqueous phase containing the thiamin. However, there was very little undissolved material after peptic digestion of the minced foodstuffs, and any error from this source may be safely neglected. Rather more solid matter remained after digestion with takadiastase and this may cause a small error in method *A*.

Thiamin determinations were also made on several of the dried materials, since, if there were no destruction of the thiamin during drying, this method would afford a more convenient way of determining the thiamin content of the food. This method is referred to as method *C* and was exactly the same as used for white flour by Slater[9] (incubation with takadiastase for sixteen hours).

Results.

The results obtained on the same sample by the various methods are shown in Table I.

TABLE I.

Thiamin Content of Minced Foods as Determined by the Thiochrome Method with Different Methods of Extraction.

Number of Test.	Thiamin. (Microgrammes per Gramme of Minced Food.)		
	Method *A*.	Method *B*.	Method *C*.[1]
I	0·66	0·66	—
II	0·38	—	—
III	0·58	—	—
IV	0·43	0·57	0·48
V	0·45	—	—
VI	0·73	0·65	0·65
VII	0·55	0·54	—
VIII	0·97	0·76	0·60
IX	0·76	0·74	—
X	0·42	0·41	—
XI	1·16	1·03	0·64
XII	1·01	0·70	—
XIII	0·53	0·50	—
XIV	0·45	0·40	—
XV	0·58	0·55	0·45
XVI	0·50	0·50	0·58
XVII	0·70	0·71	0·23
XVIII	0·67	0·64	0·55

[1] Results recalculated on wet basis.

Table I shows that with the exception of numbers IV, VIII and XII (apparently due to isolated errors in technique), the agreement between results obtained by methods *A* and *B* was close. Method *A* gave figures which averaged 5% greater than those obtained by method *B*, and this may have been due to the volume occupied by solid matter after incubation in method *A*.

Usually method *A* gave a thiochrome solution free from interfering fluorescent substances, whereas solutions obtained by method *B* always contained a small amount of white fluorescence in both the blank and the oxidized solutions. However, when beetroot was present in the diet, takadiastase did not destroy the red colour of the aqueous food extract, and a considerable interfering fluorescence made matching in method *A* very difficult. Heating with 0·1 N hydrochloric acid on the water bath, as in method *B*, destroyed the red colour and also the interfering fluorescence.

Method *B* is preferred, and results obtained by this method have been used subsequently in this paper, except where method *A* only was used on the minced food. The result obtained by method *A* was also used for test number IV.

Table II shows the percentage recovery of thiamin added (in the form of a solution) to the minced food before treatment. Method *B* was used.

TABLE II.

Percentage Recovery of Thiamin Added to Foodstuff.

Number of Test.	Thiamin Content of Foodstuff. (Microgrammes per Gramme.)	Thiamin Added. (Microgrammes per Gramme.)	Thiamin Recovered. (Microgrammes per Gramme.)	Percentage Recovery of Added Thiamin.
VIII	0·76	1·07	1·08	101
IX	0·74	1·12	1·00	90
X	0·41	1·17	1·08	92
XI	1·03	1·17	1·31	112
XII	0·70	1·20	1·16	97
XIII	0·50	1·20	1·10	92
XIV	0·40	1·20	1·26	105
XV	0·55	1·20	1·15	96
XVI	0·50	1·20	1·37	114
XVII	0·71	1·20	1·30	109
XVIII	0·64	1·20	1·29	107

Mean: 101%.

Quantitative recovery was obtained; this showed that the heating and incubation under the conditions of the test destroy no thiamin.

Method *C* is apparently quite unsatisfactory. In some cases (Tests IV, VI and XVI) there was no destruction of the vitamin; but in others destruction varied from 14% to 70%.

The following determinations were carried out on the oven-dried material.

The residual water was determined by toluene distillation on 30 gramme samples. Ash was determined on five gramme samples by heating to constant weight in a muffle furnace at a dull red heat. Fat was determined by the Rose-Gottlieb method on two gramme samples after hydrolysis of the carbohydrate by heating with 10% hydrochloric acid solution in a boiling water bath for forty-five minutes (compare Wardlaw and White[11]). Protein was determined on 0·5 gramme samples by Kjeldahl digestion with 20 cubic centimetres of concentrated sulphuric acid and a little selenium as catalyst. The factor 6·25 was used to convert nitrogen values to protein. Carbohydrate was determined by the differences between the total solids of the samples and the sum of the principal constituents, and the Calories were calculated from the amounts of fat, carbohydrate and protein in the diet by means of the factors 9·3, 4·1 and 4·1.

Table III gives the constants used in the calculation of the contribution of the butter and milk.

TABLE III.

Constants for Butter and Milk.

Constituent.	Butter.	Milk.
Ash (percentage)	0	0·73
Fat (percentage)	85·1	3·7
Protein (percentage)	0	3·5
Carbohydrate (percentage)	0	4·4
Thiamin (microgrammes per gramme)	0	0·4

The full set of results is shown in Table IV.

The water content of the minced food ranged between 56·3% and 77·7% with a mean of 66·5%. These figures are

TABLE IV.

Thiamin, Water, Ash, Protein, Fat, Carbohydrate and Calorific Content of Food Samples.

Number of Test.	Date.	Weight of Food Before Mincing. (Grammes.)	Weight of Butter. (Grammes.)	Weight of Milk. (Grammes.)	Thiamin.					Water Content of Minced Foods. (Percentage.)	Total Ash. (Grammes.)	Total Protein. (Grammes.)	Total Fat. (Grammes.)	Total Carbohydrate. (Grammes.)	Total Calories.	Non-fat Calories.	Micrograms of Thiamin to Non-fat Calories.
					Microgramme per Gramme in Minced Food.	Micrograms in Minced Food.	Micrograms in Milk.	Total Micrograms.	Total International Units.								
I	7/5/41	1,206	45	90	0·66	796	36	832	277	63·5	16	101	109	269	2,530	1,518	0·55
II	8/5/41	1,214	25	117	0·38	461	47	508	169	72·8	19	98	70	179	1,792	1,137	0·45
III	12/5/41	1,061	44	138	0·58	615	55	670	224	71·1	12	83	84	182	1,870	1,088	0·62
IV	13/5/41	1,414	35	124	0·43	610	50	660	220	68·6	16	95	125	255	2,595	1,435	0·46
V	14/5/41	1,101	46	103	0·45	496	41	537	179	62·8	13	89	119	240	2,462	1,350	0·40
VI	15/5/41	1,242	39	134	0·65	807	54	861	287	67·9	13	82	95	260	2,283	1,402	0·61
VII	19/5/41	1,219	77	254	0·54	658	102	760	254	65·5	14	86	144	273	2,812	1,472	0·52
VIII	20/5/41	1,733	42	100	0·76	1,317	40	1,357	452	71·2	27	111	121	288	2,764	1,637	0·83
IX	21/5/41	1,273	48	134	0·74	942	54	996	332	64·4	16	133	145	217	2,790	1,440	0·69
X	22/5/41	1,548	43	190	0·41	635	76	711	237	67·5	15	132	119	302	2,883	1,780	0·40
XI	26/5/41	1,141	64	155	1·03	1,175	62	1,237	412	56·3	22	145	138	268	2,975	1,692	0·73
XII	27/5/41	1,184	61	178	0·70	829	71	900	300	63·4	14	99	154	242	2,826	1,398	0·64
XIII	28/5/41	1,054	51	134	0·50	527	54	581	194	67·0	8	84	88	227	2,096	1,275	0·46
XIV	29/5/41	1,307	52	147	0·40	523	59	582	194	64·5	11	109	114	292	2,712	1,648	0·35
XV	2/6/41	1,065	44	103	0·55	586	41	627	209	66·2	13	102	91	203	2,094	1,250	0·50
XVI	3/6/41	1,274	56	126	0·50	637	50	687	229	65·1	11	126	128	244	2,706	1,518	0·45
XVII	4/6/41	1,303	48	135	0·71	926	54	980	327	77·7	7	65	76	200	1,788	1,088	0·90
XVIII	5/6/41	1,194	53	119	0·64	764	48	812	271	61·1	11	87	140	287	2,831	1,533	0·53
Mean	.. ..	1,252	48	138	0·59	740	55	795	265	66·5	14	101	114	249	2,490	1,428	0·56

similar to those obtained by Wardlaw and White[9] on the diet of Australian schoolboys—namely, 55·5 to 74·2 with a mean of 65·1.

Comparison of the Experimental Results with Figures Calculated from the Amount of Food Purchases and with the Average Australian Diet.

A complete list of the amounts of food purchased over fourteen days (May 1 to May 14 inclusive) and a statement of the average number of men to each meal were supplied. By means of this information, it was possible to calculate the average daily consumption per man. This is shown in Table V, which also includes the average daily consumption of food per "adult male" in New South Wales as determined by the Commonwealth Advisory Council on Nutrition.[11]

The person responsible for collecting the food used in the experiment ate neither porridge nor pre-cooked cereals for breakfast. It has previously been mentioned that this choice was the only one likely to influence the amount of thiamin consumed. Consequently the amounts of protein, fat *et cetera* present in a plate of porridge (including milk and sugar) and in a plate of cereal have been added separately to the experimental results. These appear in Table VI, which also includes the calculated amounts of the various constituents and the average New South Wales consumption as determined by the Commonwealth Advisory Council on Nutrition.[11] The results of Slater and Rial[12] have been used for the calculation of the nutritive value of the porridge and cereal.

TABLE V.

Consumption of Foods Compared with the Average New South Wales Diet.

Item.	Consumption in Grammes per Day.	
	Training Centre per Man.	Average in N.S.W. per "Adult Male".
Bread	211	302
Flour	28	35
Cereals	30	46
Meat	323	238
Bacon	12	6
Fish	24	24
Milk	371	512
Butter	53	58
Eggs	28	36
Potatoes	176	194
Green vegetables	139	204
Root crops	65	60
Fresh fruit	107	202
Dried fruit	3	—
Sugar	75	90

TABLE VI.

	The Training Centre Diet.				Average N.S.W. Diet-Values Obtained by Calculation.
	Values Obtained by Laboratory Analysis.	Values Obtained *plus* Plate of Porridge.	Values Obtained *plus* Plate of Cereal.	Values Obtained by Calculation.	
Total weight of food (grammes) ..	1,438	1,664	1,589	1,645	2,180
Butter (grammes)	48	48	48	53	58
Milk (grammes)	138	194	254	371	530
Thiamin (milligrammes)	0·795	0·958	0·847	0·960	1·23
Thiamin (international units)	265	319	282	327	410
Protein (grammes)	101	105	107	90	113
Carbohydrate (grammes)	249	286	285	305	494
Fat (grammes)	114	119	119	133	145
Calories	2,490	2,704	2,705	2,856	3,836
Ratio of thiamin in micrograms to non-fat Calories	0·56	0·60	0·53	0·59	0·49

The difference between the amount of milk received for analysis and the calculated amount is due to the fact that only the milk drunk as such or used in tea *et cetera* was collected separately. The balance of the milk represents that used in cooking.

The values given in the last two columns of Table VI were derived from data from a number of sources. The thiamin was calculated by means of the figures of Clements *et alii*,[9] with the exception of that for white bread. For this food the figure adopted by the Nutrition Committee of the National Health and Medical Research Council was used.[10] Protein, fat, carbohydrate and Calories were calculated by means of figures taken from the tables given in the fifth and final reports of the Commonwealth Advisory Council on Nutrition.[11,12]

In the calculation of the figures in the fifth column of Table VI, corrections were applied for the amount of inedible material according to the tables given in the fifth report of the Commonwealth Advisory Council on Nutrition;[12] but no correction was made for wastage of edible material. This was the procedure used in the investigation reported in the final report of the council.

Discussion.

Table IV indicates that the amounts of the constituents in the food received showed considerable variation from day to day. Thiamin varied from 0·508 to 1·357 milligrammes, ash from 7 to 27 grammes, protein from 65 to 133 grammes, fat from 70 to 154 grammes, carbohydrate from 179 to 302 grammes and Calories from 1,788 to 2,975 (these values have not been corrected for the pre-cooked cereal and porridge). Table VI shows a close agreement between the values obtained by laboratory analysis and those obtained by calculation when due allowance has been made for the plate of porridge or pre-cooked cereal. Almost exactly equal total weights of porridge and pre-cooked cereals were consumed, and 300 international units can be taken as the average thiamin consumption. With the exception of protein, the experimental results are a little lower than the calculated results. The figure for thiamin is 7% lower than the calculated figure, that for protein 18% higher, that for carbohydrate 6% lower, that for fat 11% lower and that for Calories 5% lower. These figures (with the exception of the protein) are of the same order of magnitude as the usual wastage in an institution. The Commonwealth Advisory Council on Nutrition[12] reported 10·6% and 5·9% wastage of edible material in a university college and an institution respectively. The agreement between experimental and calculated thiamin values is remarkably close when the errors involved in the determination of a calculated thiamin intake are considered. In the light of these results it would appear that the average New South Wales intake of thiamin as calculated by Clements *et alii*[9] is close to the true average intake.

The quantities of all constituents are lower than in the average New South Wales diet. This is particularly noticeable in the case of carbohydrate due to the lower consumption of bread, flour, cereals and sugar. Consequently, the calorific value of the diet is considerably less than that of the average New South Wales diet.

The total thiamin content in the diet examined is lower than that furnished by the average New South Wales diet; but owing to the smaller carbohydrate content, the ratio of thiamin to non-fat Calories (0·56) is slightly higher. R. R. Williams and Spies[6] consider that a value of 0·30 for this ratio is sufficient to prevent beriberi, but the optimal ratio is not known. They calculated that the average American diet supplied 0·9 milligramme of thiamin per day, with a ratio of thiamin to non-fat Calories of 0·54. These figures are practically the same as those obtained in this investigation. Slater and Rial[13] found that a ratio of 0·44 was sufficient for the normal growth and behaviour of breast-fed infants.

Many investigators have estimated the intake of thiamin necessary for adequate nutrition. The majority of these estimates vary between 300 international units (0·9 milligramme) and 400 international units (1·2 milligrammes). Recent work by Wang and Yudkin[14] and by R. D. Williams *et alii*[15] on the level of thiamin intake necessary to maintain tissue saturation, supports these figures.

Table VI shows that the choice of pre-cooked cereal or porridge for breakfast has an important influence on the amount of thiamin consumed. Oatmeal porridge is an excellent source of thiamin, but pre-cooked cereals are extremely low in this vitamin and practically all the thiamin in a plate of cereal is derived from the milk used.

The calorific intake is surprisingly low. The Commonwealth Advisory Council on Nutrition[11] regarded 3,000 Calories gross as a threshold figure, considering all diets which failed to reach this standard as subliminal diets. It is usually considered that the net calorific intake is 10% less than the gross intake—that is, 2,700 net Calories would be the adequate figure. The net calorific value of the diet as determined in the laboratory was almost equal to this figure (2,704 Calories), while the gross Calories, as calculated from the food purchases, were lower than the threshold figure. Wardlaw and White[5] found a net intake of 2,707 Calories for eighteen year old schoolboys. The League of Nations[16] considers that 2,400 Calories net should be taken as the "basic requirement" for the sedentary man. For moderate work an extra 75 to 150 Calories per hour should be added. The men studied in the present investigation were engaged in light, moderate work for about six hours a day. This would bring the requirement up to about 3,000 Calories net or 3,300 Calories gross.

However, the men showed no signs of an inadequate calorific intake, and had increased in weight while receiving the diet. It therefore seems probable that they made up the deficit of approximately 300 Calories by eating food other than that provided by the regular meals. Further inquiries revealed that approximately 21 grammes of sweets per man were purchased at the training centre each day. These would provide about 89 Calories, 72 in the non-fat form. The consumption of alcoholic beverages (which contain little or no thiamin) would provide further non-fat Calories. It can be calculated from information in the "Official Year Book of the Commonwealth of Australia" that the average consumption of alcohol by the whole Australian population (male and female) over the age of eighteen years is 13·1 grammes per day, which would provide approximately 92 Calories in a non-fat form. (The usual figure taken for the calorific value of alcohol is seven Calories per gramme. If alcohol is drunk rapidly, however, some is not oxidized, but is eliminated in the breath and urine. However, this error may be compensated by the fact that many alcoholic beverages contain protein and carbohydrate in addition to alcohol. For example, reference to the tables in the fifth report of the Commonwealth Advisory Council on Nutrition[12] shows that Australian beer of 4·2% alcoholic content has a value of 163 Calories per pound, 133 of which are derived from the alcohol.) No information could be obtained concerning the amount of alcohol consumed by the men; but as they were all young, it was probably considerably above the Australian average. One half-pint of beer (5% alcoholic content) will provide 99 Calories.

On the assumption that the men consumed 300 non-fat Calories in the form of sweets and alcohol, containing no thiamin, the average ratio of thiamin to non-fat Calories would become 0·47.

It is clear that the food consumed by the men contained sufficient thiamin to prevent an outbreak of beriberi. It is also apparent that the food supplied to the men at meals contained about the same amount of thiamin in proportion to the non-fat Calories as the average Australian or United States of America diet. But food such as confectionery or alcohol consumed between meals would seriously reduce this ratio. This reduction would be much greater in some instances than in others, and might bring the ratio of thiamin to non-fat Calories down to a dangerous level.

Summary.

1. A procedure for determining the thiamin content of a diet in the laboratory by the thiochrome method has been

described. The method adopted was to apply the procedure of Pyke to the wet minced foodstuff.

2. In order to express the result as the ratio of thiamin to non-fat Calories, determinations have also been made of ash, protein, fat and carbohydrate (the last-mentioned by difference).

3. The average daily intake per man was found to be as follows: thiamin 0·9 milligramme (300 international units), protein 106 grammes, carbohydrate 286 grammes, fat 119 grammes, Calories 2,704. The ratio of thiamin to non-fat Calories was 0·56.

4. The agreement between the values obtained by laboratory methods and those calculated from the food purchases was close.

5. The quantities of all constituents were less than contained in the average New South Wales diet; the carbohydrate intake was particularly low and the total calorific intake was about 10% less than that recommended by the League of Nations for men engaged in moderate work.

6. It is believed that the deficiency of Calories was made up by the ingestion of food other than that provided by the regular meals. We have evidence to support the belief that these extra foods were carbohydrates poor in thiamin and consequently the average ratio of thiamin to non-fat Calories might be reduced to about 0·47.

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Reviews.

STATISTICS AND ITS LITERATURE.

AN unusual book entitled "The Second Yearbook of Research and Statistical Methodology Books and Reviews" comes from America.[1] It has been edited by Oscar Krisen Buros. It is really a continuation of a scheme which was started a few years ago with the publication of the first volume entitled "Research and Statistical Methodology Books and Reviews of 1933-1938". The book is unusual because it consists entirely of quotations from scientific journals and reviews appearing in them dealing with books on research and statistical methodology. The reviews in the first volume appeared in journals covering accountancy, actuarial mathematics, agriculture, biology, botany, business, economics, eugenics, forestry, general science, mathematics, medicine, philosophy, physics, political science, public health, sociology and statistics. One of the first sections in the book is a list of the cooperating journals. In the present volume appearing under the new title, excerpts are quoted from 1,652 reviews appearing in 283 journals.

The editor points out at the beginning of his preface that tremendous advances in statistical theory have been made during the past twenty years. Coincident with this rapid development, the application of statistical technique has been proceeding with increasing speed to numerous fields in which statistical methods were formerly thought to be neither necessary nor appropriate. During the last ten years an enormous number of text-books have been published for students beginning the study of statistics. Buros goes on to state that the authors of many of these books were obviously ignorant of the revolutionary changes which have taken place in the field in which they presumed to write, and that even today text-books which present incorrect, inefficient and obsolete methods of statistical analysis continue to be published and may even be more widely used than text-books which are reasonably abreast of modern developments. It is obviously no use to tell people that many books appearing on the subject are worthless. Some assistance must be given in the selection of modern text-books, and some information must be available concerning the merits and the limitations of text-books. Buros points out further difficulties. He states first of all that books are frequently reviewed by persons who seem to know little about the subject on which they express opinions, and secondly, that most reviews by really competent reviewers are in inaccessible journals. Here, then, is the object of his book. At the risk of appearing tedious, and because the subject is without doubt of importance, we quote *in extenso* the following objectives of the editor's series of books:

> (*a*) To make students and teachers of statistics more keenly aware of the inadequacy of much of what is now presented in textbooks and classes despite the fact that such statistical techniques are incorrect, inefficient, and obsolete. (*b*) To help students, teachers, and librarians to select textbooks with greater discrimination. (*c*) To point out to students and teachers the weak and strong points of particular books. (*d*) To assist more advanced students in keeping abreast of modern developments in monograph and textbook writing and criticism. (*e*) To encourage research workers to consider and examine methodology books intended for workers in other fields and also books on general history of science, scientific method, and the social relations of science. (*f*) To emphasize that there are usually marked differences of opinion even among the more advanced students of statistical theory in their appraisal of a particular book. (*g*) To indicate the vast extension of fields in which statistical techniques are being found useful and necessary. (*h*) To discourage the writing and publication of stereotyped textbooks written by persons ignorant of modern developments in statistical theory. (*i*) To make readily available important and provocative statements which, though appearing in book reviews, have considerable value entirely apart from a consideration of the book under review. (*j*) To improve the quality of reviews by stimulating editors to take greater pains to choose competent reviewers who have the industry and the courage to contribute frankly critical reviews following a careful study of the book being reviewed. (*k*) To improve the quality of book reviews by stimulating reviewers "to take their responsibilities more seriously" by refusing to review books which they cannot, or will not, appraise competently and honestly.

[1] "The Second Yearbook of Research and Statistical Methodology Books and Reviews", edited by Oscar K. Buros; 1941. Highland Park: The Gryphon Press. Imperial 8vo, pp. 403. Price: $5.0 net.

The editor is naturally anxious about the fate of this book. Of its value to teachers and librarians there can be no doubt. Unfortunately it is not the type of book that will call for a large sale. He hopes that in future, if funds permit, he will be able to do reviews from various journals and that he will be able to introduce a section devoted to non-critical abstracts of the literature on research and statistical methodology published in the English language throughout the world. Later on he thinks that this section could be further expanded to include more important articles written in French, German, Italian, Russian, Swedish and other foreign languages. The third improvement, which could be made if sufficient help and money were forthcoming, is the publication of original criticisms of articles and papers in the periodical literature. We trust that this venture will meet with success. It is surely worthy of endowment by some far-seeing scientific body with money, if such a body exists.

PUBLIC HEALTH PROBLEMS.

IN his small book entitled "Practical Public Health Problems" Sir William Savage has made no attempt to provide a text-book.[1] He has written purely for those who already have a knowledge and practical experience of public health work, the medical officers of health and their inspectors and engineers. After a long experience in this work, he has selected a number of the important and difficult problems he has met and set down his views on them. Many of these are inadequately discussed in text-books. Some of them are usually left to the health inspector or sanitary engineer, but the medical officer of health should be the authority on them all. The practice set out in the book, of course, conforms to English laws and conditions, which in a few matters differ from our own. In all, however, the same principles of action apply. Sir William has obviously had an extensive experience, and his opinion is always interesting. His book provides a useful refresher course for all who are engaged in public health work, whether as medical officers, inspectors or engineers.

The discussion of the disposal of sewage and the control of water supplies deals only with the smaller systems such as are found in our country districts. The disposal of industrial wastes, particularly milk wastes, is dealt with at some length. This chapter may be of real value to medical officers and health inspectors in our dairying districts. The disposal of milk waste is not an easy matter. Readers unfamiliar with the problem will be surprised to read that in their detrimental effect upon river water milk and whey are each at least 150 times as harmful as ordinary sewage. A butter or cheese factory loses about 0·5% of the total milk handled. This may amount to many gallons in a day and its disposal creates a very real problem. Sir William discusses the problem in a most practical way and in considerable detail. He then describes the diseases spread by milk: tuberculosis; streptococcal, staphylococcal and salmonella infections; diphtheria and the typhoid group; and he discusses the methods of investigating an outbreak of disease spread by milk. The control of the dairy and pasteurization receive considerable attention.

A short chapter on food inspection is primarily an application of the relevant British Public Health Law, but though our law may differ a little, our problems are essentially the same, and the section will not be devoid of interest to food inspectors. The investigation of an outbreak of food poisoning is dealt with at length. In Australia this often falls to the lot of the private practitioner, or is neglected entirely, for food poisoning is not a notifiable disease, as it has recently been made in England. A chapter on the inspection of canned foods, and the recognition and significance of the "blown" tin, the damaged or leaking tin and the several types of "springer" tin is of real interest, perhaps even more to doctors' wives than to the majority of doctors.

Finally housing inspection and public health disinfection are dealt with briefly but with admirable good sense. It is good to read a vigorous denunciation of gaseous disinfection against such common organisms as those of scarlet fever and tuberculosis, and to find diseases placed in well-defined groups according to the viability of the organism and the consequent need for disinfection.

Sir William Savage has obviously written after a long practical experience of public health work. He has not attempted to be exhaustive either in the treatment of any one subject or in the sum of topics discussed. This book is of little use therefore to the reader who seeks an outline of public health practice, but it contains much to interest and stimulate those medical officers and laymen who, already trained, are engaged in this work day by day.

[1] "Practical Public Health Problems", by Sir William Savage, B.Sc., M.D.; 1941. London: J. and A. Churchill Limited. Large crown 8vo, pp. 205, with three diagrams. Price: 10s. 6d. net.

EARLY AUSTRALIAN HISTORY TOLD IN A NOVEL.

MOST Australians will probably agree that those who are born, live and die in this country, should know something of its early history, and yet it would be safe to venture the statement that not one Australian in ten has more than the most superficial knowledge of what happened in the early days of the settlement round the shores of Port Jackson, what tremendous difficulties the early settlers had to face, and how ill equipped they were to meet them. To study history sounds dull and dreary to those who have no bent in that direction; to their aid comes the historical novel. If such a book is written with intelligence and insight, those who shy at history may, if they in their turn will display a little intelligence, fill up many gaps in their knowledge. To such folk Eleanor Dark, of Katoomba, New South Wales, offers "The Timeless Land", which gives a vivid and convincing picture of the first five years of English settlement in Australia under Governor Arthur Phillip.[1] To introduce "The Timeless Land" in this way may not be quite fair, for it is one of the most notable books that have been written about early Australian history. Its standard is so high that it will be valued not only by the historian, who will find the facts as he knows them chosen and arranged with care, clothed with imagination and set out with judgement, but also by the lover of the English language for the quality of the prose.

The story is moving and dramatic. Arthur Phillip, the Governor, arrived in Port Jackson with a handful of soldiers and a number of convicts to found a new colony. With him came few, if any, skilled workmen of the type that he needed. He came to a place that "did not welcome you . . . did not look particularly fertile, and . . . was certainly not languorous". It did not "repel you . . .; it offered no enmity, no resistance. It simply waited". It was the "timeless land". The author quotes from letters and official documents, and indeed must have spent an enormous amount of time in patient research. She depicts Arthur Phillip as a man of high moral character, of courage and endurance, endowed with humanitarian instincts, all of which he undoubtedly was. She also builds up on the basis of his unshakeable faith in the destiny of his colony (for it was peculiarly his) a conception of him which few will have the temerity to question. When we read of his stupendous tasks and of his disappointments, of the failure of the Home authorities to meet his needs, we marvel at the outcome. Threatened with starvation and faced almost continually with shortage of food, Phillip displayed amazing courage. Disease took its toll of the settlers and of the natives. We read of White, the chief surgeon, and of his assistants Balmain and Arndell, and of how they struggled to maintain the health of those in their charge. We read of smallpox among the natives and of their difficulties with food supplies because the white man had come.

Mrs. Dark has studied the aboriginal, his beliefs and his customs, and has given us a picture of the working of his mind, his reaction to the invasion of his land, his feelings about the convicts who lived laborious days and had to submit to stern discipline, and about the soldiers who lorded it over them. The aboriginal must have thought about the strangeness of what he saw going on before his eyes. He had an elaborate tribal system and communal laws which he carefully observed; they must have meant something to him; he must have been able to think. Mrs. Dark's ideas of what he thought are at least logical. In any case we cannot believe that the aboriginal saw and did not wonder, and we know that virtue, loyalty, faith and steadfastness are not confined to the white races of the earth. Many readers will surely find the psychological picture of the aboriginal one of the most attractive features of the book.

Many of the native characters in the book are historical. Bennilong, though he ended in disaster, is most attractively portrayed. Other characters, an escaped convict who in death made a success of life, and an English family, Mannion by name, are fictitious.

This book will live. It is already a success. We would that those Australians who read it could gather from it some of Phillip's faith in the destiny of their country and some of his willingness to work for it.

[1] "The Timeless Land", by Eleanor Dark; 1941. London: Collins; Sydney: Angus and Robertson Limited. Demy 8vo, pp. 449. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, JANUARY 31, 1942.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE BRANCHES AS DEMOCRATIC INSTITUTIONS.

ALTHOUGH the annual meetings of only two of the Australian Branches of the British Medical Association are held in December of each year, the new office bearers taking up their duties in the new year, it is convenient for several reasons to think of Branch activities in terms of the calendar year. One of these reasons is that in the hotter months there is a gap in the monthly scientific meetings, and this seems to give a fresh impetus to all that follows. The time is therefore suitable for a discussion on the attitude of members to a body to which they should be proud to belong and whose honour and well-being they should cherish. The British Medical Association has been described as a democratic institution, and this in fact it is. An organization capable of being described as a democratic institution must have certain characteristics and its members certain privileges and responsibilities. These will be the better appreciated if we have a proper idea of what democracy itself means. As a matter of fact these are days when the word democracy is on everybody's lips, and beyond all consideration of the British Medical Association, there is a danger that, becoming a catchword, it may lose its true significance.

The word is from the Greek δημοκράτια, meaning a popular government, and derived from the two words δῆμος, the people, and κρατεῖν, to rule or govern. Abraham Lincoln's phrase "government of the people, by the people, for the people" is probably as good a definition of democracy as can be had. But mere definition is not enough. We all know that there is no real democracy in the present-day world. The principle of democracy rests on the doctrine of the equality of man. This doctrine arose from the early Christian conception of the equality of all men before God; it was applied to the political sphere and to the general social structure by Rousseau, whose writings had far-reaching effects. In regard to physical characteristics and intellectual attainment men are not born equal—inequality is inherent in the human race. Recognition of this fact will, as J. B. S. Haldane insists, not be a blow to democracy. The inequality of man apart, it is necessary to remember that an ideal democratic society is one in which there is no privilege, whether that privilege is gained by birth as a member of a privileged family, by the possession of wealth inherited or acquired, or by some signal service performed for the State. Clearly also the democratic spirit is menaced by the existence side by side of extremes of wealth and poverty. Though no real democracy at present exists, it must be owned that we have made considerable advances on the road that leads to this ideal. The opposite of democracy is what is known as absolutism—the absolute domination by a dictator. Absolutism means that the community is ruled by the will of one man; democracy denotes rule by the will of the people. The democratic state is quite compatible with monarchy and empire, as the constitution of Britain and the British Empire clearly show. The term democracy therefore does not indicate an external form of government, but "a type of political society in which the essential power of the State is wielded by the mass of the people". In other words, it has reference to the essential character of the government. We may thus conclude that a democratic society as we know it is one in which there has been established "a substantial equality of legal rights and obligations and of social and industrial opportunity". The two most important words in this definition are "rights" and "obligations". Human nature is so constituted that unless we are careful we are all apt to forget that rights and obligations are inseparable; we are all willing to accept the former, but our memories can be regrettably short in regard to the latter. Our democratic rights need not be discussed at length; it must, however, be emphasized that our rights are also the rights of others. Whether we will it or not, we are all members one of another, and we know that if "one member suffer, all the other members suffer with it". We cannot deny our membership of the body of which we are part. "If the foot shall say, Because I am not the hand, I am not of the body; is it therefore not of the body?" Clearly the body as a whole may have certain rights, and they may appear to conflict with those of one of its members. In such circumstances, since the body must function as a whole, the rights of the individual must be put on one side—the whole is always greater than the part and the part must be subservient to the whole. And what of our obligations under a democracy? Since in a democracy the rule is that of the will of the people, the people must make known their will; those with a franchise must exercise it. To do this they must not only know what they themselves want, but they must understand the whole issues at stake. Everyone will wish to share in the achievements, the glories, of a democracy; in like fashion every person must bear a full share of blame for any ignoble act of which it may be guilty. Since men are not and never will be equal, there will always be those who will lead and others who follow; the people must choose their representatives and their leaders, and having chosen them must give them every opportunity of shaping the destiny of the community towards the goal of the common good. A great deal might be written about rights and obligations, but we must be content with the observation, trite perhaps, but necessary, that while rights need not be denied, they should always convey to the mind of the recipient a call

to effort that he by his life and works show his deserving of benefit and his willingness to return something for what he has received and in like value.

Having taken a bird's-eye view of democracy, and realizing some of its implications, we are in a position to return to our original theme of the Branches of the British Medical Association as democratic institutions. Latterly there has been indication, unfortunately not a novel occurrence, of the apathy of members to Branch affairs. The fact that only 219 members of the Victorian Branch took the trouble to vote at the recent election of members of Council is fairly typical of the response that generally follows an appeal to members of the Australian Branches. The late Robert H. Todd used to say that a 30% response from an appeal to members was all that could be expected. It is only too true that those who are most negligent of their duty in the conduct of Branch affairs are generally noisiest when something is done that does not suit their particular "book". There is no need to pursue the subject much further; the parallel between a democratic State and a democratic institution should be obvious. By all means let us enjoy the rights and privileges of membership of our democratic association, but let us never forget our responsibilities to each other, to the calling that we follow, and to the larger democracy of which our democratic institution is a part.

Current Comment.

ALCOHOL AND TRAFFIC ACCIDENTS.

IN last week's issue of this journal a good deal of space was devoted to the subject of motor car driving and alcoholic intoxication. The subject came up for consideration because of three papers read at a meeting of the New South Wales Branch of the British Medical Association. It was pointed out that "driving under the influence" was part of the larger subject of motor car driving and the avoidance of accidents. Many aspects of the subject were discussed, but owing to lack of space at least two were not elaborated. The first of these is the alcoholic intoxication of pedestrians. Obviously when the consumption of alcohol is primarily responsible for an accident involving a motor car, the driver is not always to blame. The pedestrian may take too much liquor as well as the driver, and when he does he may be just as great a menace to public safety. Unfortunately the drunken pedestrian is hard to eliminate from the causes of accident. At a church in Sydney, whose rector delights in the display every week of slogans on a large canvas, there recently appeared the statement that: "The most dangerous are half-truths, half-drunks and half-Christians". With the first and last of these three we need not concern ourselves, but everyone will agree that in regard to traffic accidents the statement about "half-drunks" is quite true. The complete "drunk" is helpless, and being static, need not be considered. The present object is not to discuss ways and means of eliminating drunkenness from the public thoroughfares, desirable though it may be, but to give some indication of the extent to which consumption of alcohol by those who walk about the streets may help to cause traffic accidents. This indication is forthcoming in a report by T. A. Gonzales and A. O. Gettler, who write from New York city.[1]

Gonzales and Gettler report the results of post-mortem examinations carried out over a period of ten years after fatal traffic accidents. In these examinations they estimated the alcohol content of the brain, and they worked on the classification laid down by Gettler and Tiber in 1927. These workers used the sign "+" to denote an alcoholic brain content of 0·02% to 0·10% and held that during life the patient would be somewhat stimulated but would have no other noticeable symptom of alcoholism. The sign "++" denoted an alcoholic brain content of 0·10% to 0·25%, and the corresponding symptoms were decreased inhibitions, emotional instability, some incoordination, loss of sense of care, and so on. The sign "+++" denoted an alcoholic content of 0·25% to 0·40%, and the symptoms were unstable equilibrium, disturbed senses, slurred speech and staggering gait. The sign "++++" denoted an alcoholic content of 0·40% to 0·60%, and the symptoms were those of deep intoxication—pronounced difficulty in locomotion or alcoholic coma. In 3,471 cases the pedestrians in fatal automobile accidents numbered 2,472, and in the brain tissue of 1,637 no alcohol was found. As would be expected, of the cases in which alcohol was found, the greatest number fell into the "+++" group. The percentage of the total 2,472 in which it was shown by examination of the brain tissue that the pedestrian had been under the influence of alcohol, was 30·7. Of the total 3,471 autopsies, 214 were performed on the bodies of drivers of passenger cars, and the percentage of those who during life had been under the influence of alcohol was 54·9. Everyone will admit that these figures and percentages are not small, and it must be remembered that they have to do only with fatal accidents; the persons who received temporary or permanent injury while under the influence of alcohol are not included. Gonzales and Gettler point out that though their investigation was conducted in New York, the figures were fairly representative and should stimulate similar investigations elsewhere.

The other subject for discussion is the effect of alcohol on vision. The results of an important investigation by H. Newman and E. Fletcher have recently been published.[1] It was these two workers who devised the apparatus, mentioned in these pages last week and designed to test a subject's steering ability and the speed with which he can apply brakes on the appearance of a red light. They now report an experimental study on the relation of the alcohol concentration of the blood to driving ability, and have submitted a group of volunteers to a series of tests of vision before and after taking alcohol. The subjects included 35 men and 15 women. The basic dose was an ounce of whisky for each thirty pounds of body weight; more was given if the subject desired it, and the whole was consumed in from fifteen to thirty minutes. The object of allowing a variation of dose was to study the effect of a wide variety of blood alcohol concentrations, though it was recognized that the number of cases at any one concentration would not be large. The drinking, we are told, was done in a social atmosphere and "the drinking situation was a fairly normal one, with a minimum of tension due to the test situation". The tests were chosen so as to require the minimum of instruction and to show the least effect of practice. They had to do with depth perception, distance judgement, lateral fields of vision, eye coordination, glare resistance and glare recovery. From the results obtained it was clear that alcohol was capable of producing changes in all the components of vision tested. All the components were not affected in the same individual. Moreover, though there was an obvious tendency for persons with high blood concentrations to show more changes, this was far from true in every case. Two examples may be quoted. One subject had when tested a blood alcohol concentration of only 105 milligrammes per 100 cubic centimetres. (This is well below the proposed standard of 150 milligrammes per 100 cubic centimetres mentioned in last week's journal.) In five of the seven tests he manifested a major decrease in vision; in depth perception and in side vision he maintained his initial performances. Another subject, who had a blood concentration of 191 milligrammes per 100 cubic centimetres, showed as his only change an increase in the time of his recovery from glare. It is pointed out that

[1] *The Journal of the American Medical Association*, November 1, 1941.

[1] *The American Journal of the Medical Sciences*, November, 1941.

though in the case of the second subject there was no evidence from the tests that he was unable to drive a motor car "in the manner of an ordinarily prudent and cautious person in full possession of his faculties", he would under the standard proposed by the National Safety Council in America have stood convicted of drunken driving. The first subject, however, on account of his low blood alcohol concentration, would have the privilege, denied to the second subject, of defending himself against the charge. On the basis of other evidence he would probably have been proven guilty. Obviously determination of the blood alcohol concentration cannot be the sole criterion of intoxication.

Considering the subject of the estimation of the alcohol in the blood, one can well imagine circumstances in which it might not be beneficial in the long run to the person concerned. In a book of short stories entitled "The White-Coated Army" James Harpole has a tale of a "Juggernaut of the Road". A young man of nervous temperament has a motor car accident and is to be charged with being "drunk in charge of a motor". The family doctor, who tells the tale, is summoned to the police cells, where he finds the divisional surgeon anxious to secure a conviction. The family doctor thinks that the nervous temperament of the lad may have aggravated the symptoms of alcoholism. Eventually the divisional surgeon and the family doctor agree to stand by the result of an examination of the blood for its alcohol content, a test to which the arrested youth agrees. The finding is less than 100 milligrammes per 100 cubic centimetres of blood, and the youth is discharged. Not so very long afterwards he and a young woman are killed while he is driving home one summer evening. He had not learned his lesson. The doctor who tells the tale says: "I wish I had not suggested the blood test. I wish . . . what's the use of wishing?"

HORMONAL TEMPERATURE REGULATION.

ON exposure to a chill environment the metabolism of a cold-blooded animal falls, whereas the metabolism of a warm-blooded animal rises. The response of the poikilothermal animal is simple; it is merely the slowing of the velocity of chemical reaction with fall of temperature, for which an approximate law has been given, applicable in the animate as well as in the inanimate world. On the other hand, the rise of metabolism in a thermostatic bird or mammal subjected to cold is not so easily explained. There is, of course, voluntary muscular movement, with its liberal evolution of heat, and such exercise is much more marked in man than in the lower animal. Then there is involuntary shivering, which presents some interesting problems; for example, the total heat production, though greater than what at first might be surmized, is still far below that liberated in active muscular movement, as in running, chopping, working with pick and shovel, playing tennis and the like; further, it may be observed that the most violent shivering in the human occurs when the chilled body is exposed to warmth and danger from continued fall of temperature is over. Despite these limitations, shivering remains the chief reflex response of the homoiothermal animal to cold. The greatest problem of all is the rise of metabolism not proceeding from muscular contraction, but seemingly dispersed throughout the body wherever there is chemical change, and all chemical transformations in the living body are apparently exothermic. One thinks of hormonal control straight away, but the question which hormone or hormones is not so easily answered. The metabolic rise certainly suggests thyreoid activity, but just how cold should stimulate this gland has not been explained. There seems to be good evidence supporting the view that the suprarenal medulla comes into play partly by exsanguinating the skin and thereby diminishing the heat loss, and partly by increase of muscular tone, which does not necessarily produce visible contraction.

The latest investigation of the problem, and certainly the most thorough, was carried out in the medical school of the University of Minnesota by Allan Hemingway and Starke Hathaway.[1] Some twenty short-haired dogs were trained to lie quietly in a metabolism apparatus. Shivering was measured electrically, mechanically and visually by ingenious devices, whilst metabolism, temperature, heart and respiratory rates were mechanically recorded. The outcome of the research was that shivering accounts for something over 90% of the rise of metabolism. The hormonal rise of less than 10% can obviously be of little service in the preservation of constancy of temperature when the body is exposed to cold. The authors support the suggestion put forward by Horvath, Hitchcock and Hartman[2] that hormonal response by thyreoid and suprarenal may play a greater part in acclimatization to cold than in the temperature regulation during sudden chill. The Minnesota investigators have proved their contention with the dog as animal for experiment, but one is not justified in extrapolating their results without further researches on other animals, including the human. It is common observation that some animals are prone to shivering whilst others are not. The dog and the horse shiver readily, the cat and the cow are the reverse, and until the human being is subjected to tests as rigorous as those applied to these dogs we may keep open-minded about the share of the hormones in the maintenance of human thermostasis.

ESSENTIAL HYPERTENSION.

THE volume of research, experimental and observational, devoted to essential hypertension shows as yet no abatement under the stresses of war. A large fraction of this research is concerned with the kidney as the causative agent, and pressor substances, renin, activated renin or angiotonin, hypertension and ischemin have been posulated as being liberated from the ischæmic kidney. There are, on the other hand, critics in abundance who refuse to be convinced that kidney changes precede the high arterial pressure. H. A. Schroeder and J. M. Steele[1] have advocated plurality of causation and have classified hypertension as renal, nervous, endocrine, vascular and unclassified. Professor Julius Bauer, formerly of Vienna, now of Louisiana State University, forcibly upholds the view that essential hypertension is a clinical entity and a disease *sui generis* in which the main ætiological factor is an hereditary predisposition, in other words an abnormal gene or gene-complex.[2] He denies that there is evidence of a primary causative role of the kidneys, and regards the renal arteriosclerosis as secondary. Much of Professor Bauer's argument is destructive criticism of opposing views and he is not happy in his handling of a striking clinical fact known to all physicians in the southern States of the United States of America, and that is the remarkably high incidence of hypertension in the Negro. If a gene is causative, then the same high incidence should be found in the Negro in Africa, but it is not. Professor Bauer meets this by casting doubt on observations made in Africa and by declaring that the American Negro is a mongrel with white and Indian blood intermixed! That contact with whites and the acceleration of the pace of life arising therefrom can be a source of hypertension he scouts as absurd. The author is on surer ground when he pleads that treatment should be looked on from a broad angle, that it should be general and not symptomatic and that it is better left to the experienced physician than to specialists and super-specialists singly or in succession. He barely touches on the therapeutic aspect, but hints that medicaments have chiefly a psychic value. In this connexion it is interesting to observe that medication by potassium thiocyanate has recently been recommended again, but the candid admission that there have been instances of toxic action will make the physician cautious in adopting this drug.[3]

[1] *The American Journal of Physiology*, October, 1941.
[2] *The American Journal of Physiology*, Volume CXXI, 1938, page 178.

[1] *Archives of Internal Medicine*, Volume LXIV, 1939, page 927. (See also "The Hypertensions", by A. Rae Gilchrist, *Edinburgh Medical Journal*, November, 1941, page 752.)
[2] *Acta Medica Scandinavica*, Volume CVIII, 1941, page 18.
[3] *Bulletin of the School of Medicine, University of Maryland*, October, 1941.

Abstracts from Medical Literature.

SURGERY.

Gastric Ulcer.

A. W. ALLEN AND C. E. WELCH (*Annals of Surgery*, October, 1941) deprecate the use of the term "peptic ulcer" to include both gastric and duodenal ulcer as leading to confusion. They point out that acute duodenal lesions are more frequent than gastric lesions, and consequently the standardization of treatment may lead to disaster in the less common gastric condition. They stress the difficulty of differentiating cancer of the stomach from gastric ulcer. Their study deals with 277 cases, and details of the diagnostic measures used are set out, including gastroscopy. They consider that too much stress has been laid upon the acid level as indicating the innocence of the lesion. They claim that gastric ulcer is fundamentally a surgical lesion, but that duodenal ulcer is not. They are of the opinion that partial gastrectomy should be resorted to because of the fact that cancer may simulate ulcer, and they recommend it even though the excised ulcer should prove to be benign. Immediate surgery is advocated: (*a*) if the ulcer is of short duration and the patient over fifty years of age, (*b*) if there is no free hydrochloric acid in the stomach, (*c*) if the ulcer is in the greater curvature or prepyloric region, (*d*) if the ulcer is chronic and in the lesser curvature.

Abdominal Pain in Cyclic Vomiting.

S. KARELITZ AND S. BLUMENTHAL (*Surgery*, October, 1941) discuss the differential diagnosis of acute appendicitis and illustrate their views by a series of cases. They point out the value of intravenously administered glucose in terminating an attack of vomiting in the absence of appendicitis and in improving the patient's condition if surgical intervention becomes necessary. Cyclic vomiting is frequently accompanied by abdominal pain, and acute appendicitis may begin with recurrent vomiting. The symptoms of acute appendicitis may be masked by ketosis, and ketonuria may be present in children with acute appendicitis.

Amputation through the Thigh.

D. A. WELLS AND S. L. TEITELMAN (*Surgery*, October, 1941) discuss a method of amputation under general anæsthesia through the lower and middle thirds of the thigh. A curved anterior flap is made, the incision extending at once down to the femur, and the muscles are freed from the *linea aspera*. A flat band retractor is pushed through under the bone and against this the bone is sawn through. Pressure downwards is made by means of the retractor, the vessels being compressed against the cork block placed under the thigh. The authors advise the finest ligature and suture material.

Extensive Cutaneous Burns.

R. M. TENERY (*Surgery, Gynecology and Obstetrics*, June, 1941) made a detailed study of eight patients suffering from extensive burns, with reference to the blood chemical changes, and in particular to the changes in potassium concentration. Immediately after extensive burns a condition of primary shock may develop, associated with severe pain and fright. Then follows a characteristic period of "toxæmia", most marked in the first twenty-four hours and in favourable cases passing off in about seventy-two hours. Later, infection of the burned area may produce a bacterial toxæmia. The early toxæmic period is characterized by low blood pressure, tachycardia, hyperpyrexia, anorexia, malaise, increasing lassitude, and later, maybe, delirium, coma and death. Investigations of the pathology of this stage have in recent years centred round the blood changes. These changes include a condition of hæmoconcentration in which the proportion of cells to total blood volume is raised from the normal of 40% to 45% (the hæmatocrit reading). In one case mentioned by the author, this figure rose to 71% in one hour following burns of the total body surface. This would indicate that about half the total plasma had left the circulation and passed into the damaged tissues or onto the surface. It is essential that this fluid should be replaced by plasma transfusions. Attempts to replace it wholly by saline infusions lead to such a fall in plasma protein that œdema is produced. Saline solution should, however, be administered as well as plasma, as there is found to be a constant fall in the level of plasma chlorides and an even greater fall in the plasma sodium level. Post-mortem studies have in some cases revealed enlargement of the adrenal glands with cloudy swelling of cortical cells and areas of necrosis. It may be that the chloride and sodium changes are related to this fact, as it is well known that adrenal changes, such as occur in Addison's disease, lead to similar alterations. In one of the cases recorded the administration of adrenal cortical hormone led to a rise in the plasma sodium and chloride content, but clinically the patient did not improve. Plasma potassium readings showed a slight rise in the first forty-eight hours, but not to a toxic level. Red cell potassium figures were confusing, but there appeared to be a tendency towards a fall in the first forty-eight hours. Cell hæmoglobin content also falls in this period, so it may be that some at least of the potassium change in the cell is due to intracellular dilution by swelling of the red cells. As the cell potassium content is about twenty times the plasma potassium content, it follows that whole blood potassium varies almost directly with the hæmatocrit changes. The author concludes that, though blood changes occur constantly and must receive adequate treatment, they do not wholly account for the toxic effects of burns. In spite of treatment and control of these changes, the patient may still suffer from the toxæmia. It may be that there is, as suggested by other writers, a toxic histaminoid substance produced in the burned tissues, and that this substance is the important factor in the causation of the symptoms.

Implantation Grafts for Infected Ulcers.

K. M. MARCKS (*The American Journal of Surgery*, February, 1941) states that to accelerate the healing of infected ulcerated areas the use of minute implantation grafts has proved valuable. The conditions in an infected ulcerated area are not favourable to the survival of the usual Ollier-Thiersch graft. Much time, even years, may be wasted in waiting for natural healing. Contraction, too, may be excessive. The method recommended is to cut a thin Thiersch graft and then to divide it into pieces about two square millimetres in size. These are then buried in the granulating surface; they are pushed in with the blunt end of an ordinary needle in a direction parallel with the surface of the wound. The pieces are inserted at intervals of half a centimetre. The area is covered with gauze kept moist with normal saline solution. Outer dressings are changed daily. After five days baths of 1 in 5,000 potassium permanganate solution for twenty minutes daily are commenced. In seven to ten days the grafts appear on the surface as soft areas which grow together till the whole area is covered. The whole object is to epithelialize the whole area in the shortest possible time without regard to cosmetic or functional result. If these factors need correction at a later date, the necessary regrafting can then be done on a fresh tissue base after excision of the unsuitable portion of the new epithelial covering. Several cases are reported with illustrations. Some patients had had ulcerated areas present for as long as 15 and 16 years. Healing occurred rapidly following the use of this type of grafting.

Early Operation for Acute Osteomyelitis.

J. ALBERT KEY (*Surgery*, May, 1941) puts the case for early intervention in cases of acute hæmatogenous osteomyelitis. In patients over two years of age the staphylococcus is the causal organism in about 90% of cases. In patients who are suffering from slight staphylococcal infections, such as small furuncles, a few organisms tend to enter the blood stream. In most cases these organisms are either passed out by the kidneys or are dealt with by the natural defence mechanisms of the body. But in an occasional case the organisms settle in bone and begin to multiply in this position. The staphylococcus has the ability to produce necrosis of and to invade the surrounding tissue. The breaking down of tissue leads to a toxæmia. This is aided by the production by the organism of lethal toxins. The human body has little ability to develop an immunity to the staphylococcus. There is also a tendency for this organism to enter the blood stream and multiply there, causing a septicæmia which is fatal in about 70% of cases. It also tends to cause a pyæmia with the production of metastatic abscesses. Another feature of this organism is its ability to remain dormant but alive in the tissues, and later to show a recrudescence of activity. The interior of bone contains no sensory nerves, so that it is not till the inflammatory process reaches the periosteum that local symptoms are produced. It is probable that the process has been present in most cases for some two to five days before this occurs. The non-expansile nature of the bone prevents the usual reaction of dilatation of blood vessels and hyperæmia by which the body deals with an infection. This is more serious as the absence of antibody formation throws most of the responsibility for defence on the leucocytic and cellular reactions. The inability to expand also leads to a rise of pressure around the infective focus, which encourages the introduction of bacteria into the general circulation. For all these reasons the

author favours early operation and drainage for this disease. But this should not be undertaken without consideration for the general condition of the patient. Pain should be relieved by morphine and immobilization of the limb. Dehydration should be corrected by infusions of glucose and saline solution. Toxæmia may be lessened by the administration of antitoxin. Sulphathiazole should be given. It may not affect the local process which is walled off in the bone, but it will help to deal with any organisms which have been forced into the circulation. When these measures have been taken, the patient should be operated on. Shock and hæmorrhage should be avoided by the making of plans beforehand and by quick work. No tourniquet should be used. Gentleness is essential. Whether or not pus is found under the periosteum, the bone is opened, but care is taken to try to avoid causing an increase of pressure in the medulla during the process of drilling. It may be possible to avoid drilling altogether and to use an osteotome for this purpose. The wound is sprinkled with powdered sulphathiazole and packed with "Vaseline" gauze. It is then immobilized either in plaster of Paris or by splinting, maybe with traction. Antitoxin is continued after operation, as well as the intravenous administration of fluid and blood transfusions if necessary. The author considers that much chronic osteomyelitis will be avoided and many joints saved if this careful early treatment is followed. In infants under two years of age operation is usually delayed, as the greater softness of the bone gives rise to a condition more like an abscess in soft tissues. Usually delay is recommended till an extraosseous abscess forms, and this is opened without the bone being disturbed.

Kirschner Wire Fixation of Hip Fractures.

J. B. CHESTER (*Surgery, Gynecology and Obstetrics*, November, 1941) describes in some detail a method for the fixation of fractures of the femoral neck by the insertion of a number of Kirschner wires. The author reviews briefly the other methods which have been employed, and claims that many of their disadvantages are absent from the method he favours. Premedication with a barbiturate, morphine and hyoscine is followed by the injection of a 2% solution of procaine into the hip joint and above the greater trochanter so as to block the skin just below the trochanter without obscuring the bony landmarks in the latter situation. Accurate reduction of any deformity is then performed by the ordinary methods of manipulation, checked radiographically and maintained by fixation on an orthopædic table. Five 16-gauge Kirschner wires are then inserted through the skin without an incision being made. Their direction is so arranged that they traverse the femoral neck, the fracture line and the head of the bone, and so that they make pronounced angles with each other. The accuracy of their insertion is verified by lateral and anteroposterior skiagrams, and any wires in faulty positions are removed and replaced. When the wires are seen to be satisfactorily placed, their ends are bent over at the point where they enter the bone. By pressing the skin inwards, the excess wire is cut off at a point which is estimated to be at about three-eighths of an inch below ordinary skin level. Within a few days or weeks the patients are allowed up in wheel-chairs, but no weight bearing is permitted until X-ray examination shows firm union. The wires are left in position for about eight to ten months. The author claims for this method that the crossed position of the wires holds the head firmly and that their bent-over ends prevent any tendency for the wires to travel towards the pelvis. The method is free from shock, and the author states that it has been used in the treatment of 23 patients with satisfactory results.

The Suitability of Extravasated Blood for Reinfusion.

E. W. PAGE (*Western Journal of Surgery, Obstetrics and Gynecology*, November, 1941) reports the results of a study of the composition of venous and abdominal blood in thirteen cases of ectopic pregnancy, made with reference to the suitability of extravasated blood for reinfusion. Red cell count, total and differential white cell count, hæmoglobin value, sedimentation time, clotting time, appearance of plasma and microscopic appearance of red cells were the observations made. The results are given in tabular form, and a discussion is presented of the literature on autotransfusion and of the present observations. The author concludes that reinfusion of the abdominal blood in cases of ruptured ectopic gestation may be a life-saving procedure, and, given proper precautions, no more dangerous than the use of preserved blood. The chief danger is in the use of hæmolysed blood. The red cells become increasingly fragile the longer they remain in the peritoneal cavity, and for maximum safety the interval between onset of acute symptoms and reinfusion should not exceed thirty-six hours. Hæmoglobin value and red cell counts were materially the same in venous and abdominal samples. The neutrophile cells were reduced. Clotting was indefinitely prolonged, probably due to defibrination, and the use of citrate is therefore unnecessary. The author advises that the abdominal blood, if used for reinfusion, should not be diluted, shaken, warmed or squeezed out of sponges. In the cases under review the abdominal blood was collected by suction.

Myasthenia Gravis and the Thymus.

ALFRED BLALOCK (*The American Journal of Surgery*, October, 1941) states that *myasthenia gravis* is a disease of unknown ætiology characterized by abnormal fatigability of muscles, which, with other organs, show lymphocytic infiltration. It has been found to be associated in many cases with an abnormality of the thymus gland, whether in the nature of a simple tumour (thymoma) or of a persistence of the gland due to failure of normal involution. This association is fairly constant. The author quotes Norris, who suggested that the thymic changes will be found in *myasthenia gravis* in proportion to the care exercised in looking for them. Up to 1940, 54 instances of abnormalities of the thymus gland had been reported in approximately 110 autopsies or operations on patients suffering from *myasthenia gravis*. Some abnormality of the thymus should therefore be suspected in every case of this disease. An X-ray examination, particularly with the patient in the lateral position, may demonstrate definite enlargements or tumours. The effects of removal of a thymoma in such a case are difficult to assess, firstly, because so few operations of this type have so far been performed, and secondly, because remissions are known to occur in this disease, and these confuse critical judgement of operative effects. The operation is relatively simple. The author concludes that clinically demonstrable thymomata should be removed from patients with *myasthenia gravis*.

Leucocyte Exhaustion in Intestinal Obstruction.

F. I. HARRIS AND J. S. FELDHEYM (*The American Journal of Surgery*, November, 1941) present a series of ten cases of small bowel obstruction in which daily white cell counts were made following operation. In all of these a marked leucopenia was noted in the immediate post-operative period of from one to seven days. The leucopenia was not associated with surgical sepsis and did not necessarily indicate a fatal prognosis, as six of the ten patients recovered. Stained films showed an absence of immature white cells and a relative increase in the more mature forms of neutrophile cells. The authors believe that the leucopenia observed is due to the sudden release of toxic products from the obstructed bowel, causing a temporary bone marrow paresis. They consider that the leucopenia may prove of assistance in diagnosis, and describe how help was obtained from a white cell count in one of their cases. They state that treatment should be directed towards the elimination of toxins rather than towards specific therapy, to raise the leucocytic level.

Irradiation of the Ovaries in the Metastatic Breast Carcinoma.

E. A. POHLE (*The American Journal of Surgery*, November, 1941) reports two cases of extensive metastatic breast carcinoma in which the patients were treated by the induction of an artificial menopause by irradiation of the ovaries. Their respective ages were thirty-nine and forty-seven years, and both showed considerable temporary improvement both clinically and radiographically. In both patients lung metastases disappeared, and in one bony metastases recalcified. The author advises the X-ray sterilization of all patients with metastases from breast carcinoma, regardless of age, and suggests that this measure be applied prophylactically to all women of forty years or older.

Massive Gastric Hæmorrhage.

J. V. BOHRER (*Annals of Surgery*, October, 1941), writing on massive gastric hæmorrhage, states that penetrating ulcer is the commonest cause and then cirrhosis of the liver. General management consists of the taking of frequent pulse rate and blood pressure readings, attention to shock, aspiration of blood by a tube with normal saline solution. The use of the tube does away with straining and is used for feeding. Blood transfusions are given, even though the bleeding has not been stopped. Gastrectomy is the operation of choice both for control of hæmorrhage and ultimate cure of the ulcer. Local excision of a gastric ulcer combined with gastro-enterostomy gives satisfactory results. The author recommends the use of non-absorbable suture material for plication of an ulcer or ligature of a vessel, and also the hypodermic administration of vitamins *B* and *C* during the postoperative period.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on September 10, 1941, at the Children's Hospital, Melbourne, DR. H. L. STOKES, the President, in the chair. The meeting took the form of a series of clinical demonstrations by some of the members.

Pulmonary Hydatid Cyst.

DR. J. G. WHITAKER showed a boy, aged ten years, who prior to his admission to hospital had fallen from a tree, and during a coughing attack had coughed up some blood and mucus, but nothing recognized as hydatid material. Physical signs directed attention to the base of the left lung, and skiagrams revealed the presence of a cystic structure, which was outlined faintly in the negative film. An instillation of lipiodol into the bronchial passages was made, but none of the substance entered the cystic area. Neither the Mantoux nor the Casoni test produced a reaction; but the hydatid complement fixation test produced a double positive response. Eosinophile cells amounted to 20% of the leucocytes counted in a specially stained blood film.

Dr. Whitaker commented on the variability of the radiological appearances of hydatid cysts of the lung, and considered that if an operation had to be undertaken, the two-stage packing procedure might be advisable. He added that he had decided to give the boy a month in which to have another accident, which might result in expulsion of cystic material and be followed by spontaneous cure.

DR. ROBERT SOUTHBY said that the optimal time for operation called for judgement which had to take into consideration the avoidance of secondary infection. He recalled that on one occasion on which the late Dr. Hamilton Russell had operated on a patient, the whole thoracic cavity had become filled with pus; after freezing the chest wall with ethyl chloride, he had made a stab incision in the back, through which the pus and cyst contents had escaped, and the patient made a satisfactory recovery.

DR. C. J. O. BROWN remarked that Professor H. R. Dew's well-known book was written in the early days of the use of lipiodol in radiography. Dr. Brown said that by means of iodized oil preparations it had been demonstrated that 80% of pulmonary hydatid cysts ruptured into the bronchial tree, but that some or all of the membrane was left behind; this caused low-grade infection, atelectasis and disorganization of the lobe with chronic pulmonary suppuration, or else débris remained and lung abscess supervened, or attacks of recurrent spitting and coughing followed. A ramifying epithelial-lined cavity formed with bronchial tubes emptying into it. Dr. Brown was by no means optimistic about the so-called "spontaneous cure" of hydatid cysts of the lung.

Dr. Whitaker had said that in the present case there was no fluid level and no fluid was left, so there must be free communication and no valvular opening. There was no response to the Casoni test, though the complement fixation test produced a positive reaction; actually there was not much proof that they were dealing with a hydatid cyst. The iodized oil had not entered the cavity; this was probably due to bad filling; but Dr. Whitaker suggested that it would be worth while to inject the oil directly into the cyst, and if it was a clean-walled cyst he did not think that an operation was indicated.

DR. H. DOUGLAS STEPHENS expressed dissatisfaction with the Casoni tests carried out at the hospital. He doubted whether the fluid used was suitable; negative results were frequently obtained when he confidently expected positive ones.

Dr. Whitaker, in reply, thanked Dr. Brown particularly for his interesting remarks. Dr. Whitaker had not considered, before he heard Dr. Brown's views, that the cyst might be a ruptured one partially filled with air; he had been thinking that he was dealing with hæmorrhage and an unruptured cyst. He pointed out that Professor Dew was not giving his own statistics, but quoting other authorities, when he used the figure 80%.

Diaphragmatic Hernia with Microcytic Anæmia.

Dr. Whitaker also showed a boy, aged fourteen years, whose case had aroused great interest at the meeting of the society held on June 11, 1941, when presented by Dr. Mona Blanch (see THE MEDICAL JOURNAL OF AUSTRALIA, September 13, 1941, page 306). The boy had come into Dr. Whitaker's care because of a suggestion by Dr. D. M. Embelton that Dr. Whitaker should try to fix the stomach in a further attempt to overcome the herniation. He had performed laparotomy and had had no difficulty in pulling the stomach out from what seemed to be a paracesophageal hernia. He had been unable to define any aperture in the diaphragm itself. The stomach was sewn to the abdominal wall and gastrostomy was carried out at the cardia. The after-result was disappointing, as the herniation was still present and the state of affairs unaltered. Filling with radio-opaque material introduced through a tube into the stomach had not been carried out, and Dr. Whitaker was not certain that the stomach had remained fixed to the abdominal wall and out of the sac. He particularly stressed the point that he doubted whether there was a definite hernial orifice; there was a saucer-shaped wound in the diaphragm, which did not seem capable of repair.

DR. MONA BLANCH expressed doubt as to whether the stomach was still attached to the abdominal wall. The tube, which had been inserted at the operation, had come out, and when it was reintroduced an effort made to put a barium preparation through it had failed. She suggested that the tube might lead into the peritoneal cavity walled off with adhesions; there was a large hole, but no food escaped from it.

DR. DOUGLAS STEPHENS said that he had watched Dr. Whitaker perform the operation. The stomach was plicated and the gastrostomy was performed entirely satisfactorily. It had to be remembered, however, that the stomach was an elastic organ and that tremendous suction developed in paracesophageal herniæ. The result was instructive; at the time of the operation the measures adopted gave promise of a satisfactory outcome, but apparently the operation had not proved to be successful.

DR. C. J. O. BROWN asked Dr. Whitaker whether he had seen any evidence of ulceration, hæmorrhage from which might account for the anæmia.

Dr. Whitaker, in reply, said that he had seen no evidence of ulceration, but that it would appear that bleeding occurred from the aberrant stomach mucosa. He quite agreed with Dr. Blanch that the stomach had become detached. He reiterated that he did not think a hernial orifice existed, and certainly its presence had not been proved. He regretted that he had not been able to carry the progress of the case further forward.

Bowel Anastomosis.

Dr. Whitaker then described to the meeting the management of a boy who, on the twelfth day after appendicectomy, had shown signs of obstruction of the intestine. He said that the signs had gradually become more urgent and a decision was reached to operate again. The actual state of affairs had been found to be even worse than had been anticipated, and on attempting to free the adhesions in the right iliac fossa he was faced with a denuded, torn and bleeding bowel with exposure of the muscle coat, and remained in doubt as to whether the obstruction was relieved. In those circumstances he had anastomosed the most damaged loop of bowel to the transverse colon and the boy had made a good recovery. Dr. Whitaker added that had he performed an ileostomy the patient might have been just as well off; but it had occurred to him that the report might interest some of his colleagues.

DR. B. R. HALLOWS said that the treatment of obstruction in appendicitis was an important problem and that it was always a grave decision when the surgeon felt that he had to reopen the abdomen. He was grateful to Dr. Whitaker for the suggestion made in the report of the procedure he had adopted. That procedure was little harder to carry out than the more usual drainage. Dr. Hallows felt sure that Dr. Whitaker's successful result would be of value to the other surgeons in the future.

Bony Deficiency in the Skull.

DR. B. R. HALLOWS showed a boy, aged six years and four months, who had come into his care five months after being hit on the head with a stick. A swelling had appeared, which remained for a week before softening occurred. The swelling subsided slowly during the following two weeks and then disappeared rather rapidly. After its subsidence the child's mother had noticed a depression in the right parietal bone, about one and three-quarter inches long, in the sagittal plane and about one inch wide. That depression had persisted, and it was noted on examination that a slight impulse occurred when the child coughed. Dr. Hallows regarded the original condition as a cephalhæmatoma, but considered it extremely rare in association with bony deficiency. The fact of complete deficiency of bone had been confirmed radiographically, and a specimen of the boy's blood serum had failed to yield the Wassermann reaction.

Dr. Hallows then referred to a classification of the causes of localized bony deficiencies in bones of the skull. He said that the congenital deficiencies lay mainly in the region of sutures, particularly at the pterion and asterion, or were due to delayed ossification in the region of the parietal foramina. The traumatic group included depressed fractures, hyperæmic decalcification, operative interference and pressure from tumours, including aneurysms. The inflammatory conditions might be acute or subacute, or associated with such chronic conditions as tuberculosis or syphilis of bone. Other causes might be neoplastic, constitutional or idiopathic. Dr. Hallows went on to say that it was reasonable to assume that the condition was due to hyperæmic decalcification. The history of trauma followed by a hæmatoma was present, and six weeks later there had been a rapid, almost sudden, disappearance of the swelling, perhaps because of rapid absorption of the diploæ after decalcification of the outer table of the bone. In support of that argument he referred to three case histories to be found in Greig's "Surgical Pathology of Bones", at page 136. In those cases loss of substance had been due to trauma without superadded infection.

Dr. Hallows further said that four principles had been laid down by Leriche and Policard: (*a*) If the circulation was maintained within certain limits, the bone remained unchanged. (*b*) If hyperæmia was produced, the bone underwent rarefaction, decalcification and osteoporosis. (*c*) If the blood supply was restricted, the bone underwent consolidation, increased density and osteosclerosis. (*d*) If the blood supply was cut off, the bone underwent necrosis. By the application of those principles, therefore, normality of bone was indicative of normal circulation in the bone; rarefaction indicated arterial hyperæmia; osteosclerosis suggested arterial obstruction; and bone necrosis meant arterial obliteration.

DR. J. G. WHITAKER, after thanking Dr. Hallows for drawing attention to the subject, demonstrated from a series of skiagrams the progress of events following a fracture of the skull of a girl who had been in his care. The fracture had been sustained in 1933, and the progressive films in 1940 and 1941 exhibited changes which had been interpreted as those of absorption of bone after trauma. Dr. Whitaker reported that the child recovered entirely satisfactorily in other respects.

DR. H. DOUGLAS STEPHENS said that he had seen a number of patients with congenital deficiencies of the bones of the skull; but he had not seen a lesion like that shown by Dr. Hallows. Dr. Stephens had had occasion to operate on a baby with a pond-shaped depression in the skull six weeks after its appearance; the elevators had come through soft tissues, as the whole centre was devoid of bone, and the bone had not developed later. He supposed that that deficiency was ascribable to hyperæmia also; if that explanation was correct, he would have to admit the possibility in the case of Dr. Hallows's patient.

DR. A. G. VILLIERS reminded members that, some three years earlier, Dr. Mostyn Powell had shown a case of bilateral congenital deficiency in the parietal areas and had quoted three other instances of congenital deficiency that had come under his notice.

Dr. Hallows, in reply, said that one of the difficulties was that the original trauma seemed to have been relatively trivial. He was still of an open mind concerning the underlying pathological process, and he found it difficult to convince himself that the hyperæmic state was the only factor. Dr. Southby had suggested to him the possibility that the accident had only attracted the mother's attention to a previously existing congenital deficiency.

Raised Right Hemidiaphragm.

DR. J. D. BEGG showed a girl, aged four years and six months. It had been accidentally discovered that the right side of the diaphragm was unusually elevated. On March 18, 1941, the child was admitted to the surgical ward with subacute arthritis of the left hip joint. All movements at that joint except abduction were limited. She had been in contact with a cousin suspected of having tuberculosis; the intracutaneous Mantoux test produced a positive reaction; radiographic examination of the pelvis revealed no abnormality. By March 31 the hip was freely movable and the child was discharged to her home. Dr. Begg saw her at the out-patient department on June 9. At that time she had had a cough for four weeks, with profuse production of frothy sputum. Resonance was impaired in the right axilla, but there was very little alteration of the breath sounds and no adventitiæ were noted. Dr. Begg inspected a skiagram prepared just before she left the ward, and was surprised to note that the right side of the diaphragm was raised to the level of the fourth intercostal space. When he saw the child again on June 19 she had had a fresh cold, and many râles and rhonchi were audible, particularly towards the base of the right lung. A Casoni intradermal test produced no reaction. A bronchogram was prepared on June 30, but no abnormality of the bronchial tree was detected. On July 7 no abnormal appearances of the gastro-intestinal tract were disclosed by an opaque meal investigation.

Dr. Begg said that no change had taken place in the level of the diaphragm, that the patient's condition had not altered except for the disappearance of the adventitious sounds, that she was still subject to frequent colds and that he was unable to explain the position of the diaphragm. He then went on to say that *"eventratio diaphragmatica"* had been described and named by J. L. Petit in 1750. The anomaly was nearly always left-sided; all except one out of 30 cases reported by 1923 were left-sided. From a clinical point of view the signs could be classified according to the disturbances of thoracic and abdominal organs. The thoracic disturbances included palpitation, cardiac pain, dyspnœa, cyanosis, and bronchitis or bronchopneumonia. The abdominal disturbances were flatulence, vomiting or dyspepsia due to disturbance of the stomach, and constipation or even volvulus from intestinal upset. The characteristic radiological signs were (i) that the condition was more frequently found on the left side, (ii) that the elevated diaphragm formed an unbroken bowline in the chest, (iii) that a gaseous space could be discerned at the level of the cardiac orifice, and (iv) that an inversion of the usual movement of the diaphragm in respiration occurred. Dr. Begg added that lung markings might be seen through the gaseous space below the bowline. The differential diagnosis from diaphragmatic hernia was made by observation of the irregularity of the bowline and the level of fluid above the normal position of the cardiac orifice. In diaphragmatic hernia the "cup and spill" cascade type of filling was noted during an opaque meal examination, together with the bilobed appearance of the stomach outline.

Dr. Begg then went on to consider theories of the causation of eventration. He said that as the condition had been found in the fœtus, or within a few hours or days of birth, the possibility of congenital developmental defect had to be accepted; but as it commonly appeared in adult life, it was attractive to ascribe the condition to phrenic nerve paralysis, though that raised the whole question of the innervation of the diaphragm. Other theories could be dismissed as fantastic. It was a fact that paralysis of the phrenic nerve produced similar radiological appearances. In conclusion, Dr. Begg pointed out that in the patient he had shown the anomaly was on the right side; the Mantoux test produced a positive reaction, and the child might have tuberculous glands which might conceivably be pressing on the phrenic nerve. That combination of events might account for the unusual position of the diaphragm.

DR. ROBERT SOUTHBY said that the clinical history and radiological appearances were consistent with the presence of a tumour under the right side of the diaphragm. He suggested that if hydatid cyst could be ruled out, the possibility of the presence of a dermoid cyst should not be forgotten.

DR. KEITH HALLAM drew attention to certain anatomical factors, which he considered to be in favour of a congenital origin of the anomaly. He mentioned the site of the duodenal cap, the transverse position of the stomach, the high position of the duodenum and of the hepatic flexure, and the fact that the liver was not displaced downwards. He remarked, however, that there were certain fundamental facts against that explanation. In a membranous partition the qualities of recoil and vitality of the normal diaphragm would be absent or at least inert. The child's diaphragm was a vital one and exhibited no paradoxical movement with respiration. The diaphragm contained fibrous, elastic and muscular tissues and was normal. If the eventration of the diaphragm was a congenital anomaly, the partition would be essentially a fibrous membrane, and would not show the vital functions of elastic and muscular tissue. The phrenic nerve could not be paralysed because movement of the diaphragm occurred. If the explanation was not the presence of a hydatid cyst, some other cause acting beneath the diaphragm should be sought.

Dr. Begg, in reply, said that diaphragmatic movement was diminished; it was just possible to say that the diaphragm moved. He thought that dermoid cysts were always found in the mid-line of the body; if one was present, he was not at all clear as to how he could establish the fact. On careful consideration he had formed the opinion that the paresis was due to involvement of the phrenic nerve and that recovery was possible. A point that had interested him was the frequency with which eventration had been reported so much more commonly on the left side. Perhaps it was easier to identify it on that side, and for that reason examples on the right side might not often be observed.

Chronic Pulmonary Disease.

DR. HOWARD WILLIAMS showed a boy whom he had introduced at the meeting on May 14, 1941. (See THE MEDICAL JOURNAL OF AUSTRALIA, July 5, 1941, page 18.) He said that bronchograms had been prepared, from which it had been presumed that early dilatation of bronchi and extensive collapse of the basal lobe of the lung were present. At a bronchoscopic examination a little mucopus had been obtained from the right main bronchus and slight narrowing had been noted, but no definite evidence of the presence of a foreign body was obtained. Dr. Williams said that he thought the members would be interested to know the results of those investigations, and that they might be disposed to discuss the diagnosis of atelectasis of the major portion of the basal lobe of the right lung and what treatment was possible. The boy still had a diminished air entry and some wheezing, though it was less in degree than it had been earlier. Finally, the boy was well and was gaining in weight.

Dr. Southby expressed the view that the collapse of the lung might be associated with a foreign body; but he thought that the ultimate prognosis was quite good and that there was no indication for active interference.

DR. C. J. O. BROWN said that he could not agree with Dr. Southby's prognosis. The lobe had been atelectatic for nearly a year; the condition might even have been congenital. He had seen gross bronchiectasis in young patients supervening on atelectasis of a lobe. Indeed, most patients with bronchiectasis gave the same story of repeated attacks of coughing and pneumonia, and it was usually difficult to decide whether the pneumonia led to the atelectasis or *vice versa*; the remainder of the lung tissue became emphysematous from the pull of the negative pressure. Apart from the extreme youth of the patient, Dr. Brown considered that the case was ideal for the operation of lobectomy, which in his opinion should be performed within a reasonable period. He added the comment that the patient's youth might actually be an advantage. Hyperplasia had been shown to occur in rats and young animals only; in older animals compensatory dilatation, not necessarily emphysematous, was the rule, rather than simple hyperplasia. Finally Dr. Brown said that he took it that it had been decided that the child was not tuberculous; but even without that complication, most patients with atelectatic lobes and bronchiectasis did not make satisfactory progress without successful lobectomy operations. Even after lobectomy death might occur from supervening tuberculous infection within a few years. He had known this to happen, and had noted that it was usual in all reported series of lobectomies for bronchiectasis. He felt certain that, to be well, the boy would require lobectomy sooner or later.

Dr. Whitaker said that he thought that lobectomy should be performed, but it was a nice point to be able to decide the most favourable time for it. The younger the child, the easier the operation would be. It had been performed on a younger patient at the hospital, and from the technical point of view it was found to be easier than was usual with older subjects.

Dr. Brown said that he agreed with Dr. Whitaker. On theoretical grounds, the earlier the operation was performed, the better would be the prospects for the patient. He anticipated that lobectomy would be safe and simple, and that convalescence would be trouble-free and recovery complete. He added that there might be some lingering doubt about tuberculous infection; but if that could be excluded, he thought that the removal of the lobe should be found to be as simple as it was in a dog.

National Emergency Measures.

MEDICAL EQUIPMENT.

THE Medical Equipment Control Committee desires to direct the attention of all members of the medical profession to the following information.

Economy in the Use of Drugs in War-Time.

The following list of drugs, classified in accordance with their therapeutic efficiency as well as with their availability, is presented as a war-time stimulus to their thoughtful and economical use.

The classification adopted is as follows:

"A": Essential drugs in which strict economy in use should be observed.

"B": Drugs essential for certain purposes but not for others. To be used with strict economy for their specific therapeutic and pharmaceutical purposes.

"Aus.": Drugs available from local sources. To be used where possible as substitutes for imported drugs.

"C": Drugs not considered essential and which do not justify importation or manufacture.

With the necessary restriction of imports, due in part to the strain imposed on shipping facilities, and to the need to conserve foreign exchange resources, the most rigid economy compatable with therapeutic requirements must be practised in the prescribing of non-Australian drugs. These are mainly grouped as "A" and "B".

Even with medicaments of Australian manufacture ("Aus.") an economic attitude must be adopted, that materials and skilled labour may not be needlessly diverted from other essential services.

Wherever possible the drugs have been listed and classified under the official names adopted by the British Pharmacopœia (1932) and its addenda, and by the British Pharmaceutical Codex. As some of these in later pharmacopœial addenda are as yet unfamiliar, many proprietary identities and equivalents are listed for reference.

In war-time it is more than ever desirable to order drugs by their non-proprietary names, otherwise a false impression of scarcity may be created by the disappearance of a particular brand from the market. If the prescribers have a preference for any individual proprietary, this may be indicated by bracketing the proprietary name with the official name.

No drug which could be strictly regarded as essential has been listed as "C", but it must be realized that owing to import difficulties and short supplies a few of wide use and popularity have been included under this heading. Where substitutes have appeared to be necessary, they have been indicated.

Drug.	Classification.	Substitute or Equivalent and Remarks.
Acacia	B	Mucil. Amyli. E.F.A., Decoct. Lini.
Acaprin	—	Pirevan is identical.
Acetanilidum	C	Substitutes: acetylsalicylic acid or phenacetin.
Acetarsol	A	See arsenicals (organic). Substitute for emetine in amœbic dysentery.
Acetylcholine	C	
Acetonum	B	Under Government control. For skin preparations as fat solvent use alcohol or ether.
Acidum Aceticum	Aus.	
Acidum Acetylsalicylicum	Aus.	
Acidum Ascorbicum	B	Use fruit juices and other dietetic sources where possible.
Acidum Benzoicum	A	
Acidum Boricum	B	Boric acid in boric lint is wasted. Reserve for eye lotions. In dermatological practice saline compresses may be used instead of boric fomentations, and for dusting powders boric acid may be replaced by kaolin, magnesium carb., starch, talc or zinc oxide.
Acidum Citricum	B	Reserve for use in preparation of medicinal citrates.
Acidum Chromicum (Chromii Trioxidum)	C	Pot. permang. is a partial substitute.
Acidum Glycerophosphoricum and its salts	C	No substitute necessary.
Acidum Hydrobromicum	C	Substitute bromides.
Acidum Hydrochloricum	Aus.	
Acidum Hydrocyanicum Dil.	C	No substitute necessary.
Acidum Lacticum	Aus.	
Acidum Mandelicum	A	
Acidum Nicotinicum	B	Use dietetic sources where possible.
Acidum Nitricum	C	
Acidum Oleicum	C	
Acidum Phosphoricum	C	
Acidum Salicylicum	A	
Acidum Sulphuricum	Aus.	
Acidum Tannicum	A	
Acidum Tartaricum and its salts	C	
Aconitum	C	
Acriflavina	A	Proflavine (Aus.).
Adeps Benzoinatus	C	Substitute Adeps. May be preserved by the use of chlorocresol or chlorinated xylenol—0·25%.

Drug.	Classification.	Substitute or Equivalent and Remarks.
Adeps Lanæ	Aus.	
Adrenaline and its salts	A	
Æther	Aus.	
Æthylenum	C	
Æthylis Chloridum	Aus.	
Æthylmorphinæ Hydrochloridum	C	Dionin is identical.
Agar	B	Agar should be reserved for bacteriological media.
Albucid	—	Sulphacetamide is identical. See Sulphanilamides.
Alcohol	Aus.	
Alcohol Isopropylicum	C	
Allobarbitonum	A	See Barbiturates.
Aloe	A	
Aloinum	A	
Alstonia	Aus.	To replace cinchona, calumba, gentian and other bitter tonics.
Alumen	Aus.	
Aluminii Acetas	C	
Aluminii Hydroxidum	C	Use Mist. Alum. Hydrox. E.F.A.
Amidopyrina	C	Substitutes: acetylsalicylic acid, phenacetin.
Aminophyllin	C	See Theophylline, Sodii Acetas.
Ammon. Brom.	A	Substitute sod. or pot. brom.
Ammonii Carbonas	A	
Amonii Chloridum	A	
Amonii Phosphas Acidus	C	
Amphetamina	C	See Benzedrine.
Amylis Nitris	Aus.	
Amylocainæ Hydrochloridum	B	Reserve for spinal anæsthesia.
Amylum	Aus.	
Anæsthesin	—	Benzocaine is identical.
Anethum	C	See Olea *d.* Essential oils.
Aneurinæ Hydrochloridum	B	Use dietetic sources where possible.
Aniline dyes (medicinal)	A	These include brilliant green, crystal violet (gentian violet), fuchsine, malachite green, methyl violet and scarlet red.
Anisum	C	See Olea *d.* Essential oils.
Antimonii et Potassii Tart.	B	Reserve for use in protozoal diseases.
Antimonii et Sodii Tart.	B	Reserve for use in protozoal diseases.
Apomorphinæ Hydrochloridum	A	
Areca	C	
Arecolinæ Hydrobromidum	C	Substitutes: pilocarpine and physostigmine.
Argenti Nitras	Aus.	
Argent Proteinas	Aus.	Protargol type.
Argent Proteinas Mit.	Aus.	Argyrol type.
Argyrol	—	See Argent Proteinas Mit.
Arseni Trioxidum	A	
Arsenicals (organic)—		Proprietary equivalents of arsenicals:
Acetarsol	A	Acetylarsan, Kharophen, Spirocid, Stovarsol.
Neoarsphenamine	A	Neo-Kharsivan, Neo-Salvarsan. Novarsenobillon (N.A.B.). Novarsan, Novostab.
Sodi Aminarsonas	C	Atoxyl.
Sulpharsphenamina	A	Karsulphan, Metarsenobillon, Myosalvarsan, Sulphostab.
Tryparsamidum	A	
Atebrin	—	Nepacrinæ Hydrochloridum is indentical.
Atophan	—	Cinchophenum is identical.
Atoxyl	—	Sodii Aminarsonas is identical.
Atropina and its salts	A	
Aurantii Cortex	Aus.	To replace such aromatic bitters as sepentary and gentian.
Avertin	—	Bromethol is identical.
Balsamum Peruvianum	C	
Balsamum Tolutanum	B	Reserve for Tr. Benz. Co.
Barbitonum	A	See Barbiturates.
Barbitonum Soluble	A	See Barbiturates.
Barbiturates— Group I, Official.		Proprietary equivalents:
Barbitone	A	Veronal.
Barbitone Soluble	A	Medinal. Veronal-Sodium.
Hexobarbitone	A	Evipan.
Hexobarbitone Soluble	A	Evipan Sodium.
Phemitone	A	Prominal. Rutonal.
Phenobarbitone	A	Luminal. Gardenal.
Phenobarbitone Soluble	A	Luminal Soluble, Gardenal Soluble.
Group 2, Unofficial.		
Allobarbitone B.P.C.	A	Dial.
Iso-amylethylbarbituric Acid	A	Amytal.
Pentobarbitone Sodium	A	Nembutal. Amytal Sodium.
Barii Sulphas	B	For X-ray diagnosis.
Bayer "205"	—	Suramin is identical.
Belladonnæ Folium	Aus.	
Belladonnæ Radix	Aus.	
Benzaminæ Lactas	C	Substitute Procaine Hydrochloride.
Benzedrine	—	Amphetamina is identical.
Benzocaina	A	Anæsthesin is identical.
Benzoinum	B	Reserve for Tr. Benz. Co.
Benzylis Benzoas	A	
Betainæ Hydrochloridum	C	
Betanaphthol	C	
Bismuthum Præcipitatum	B	Reserve for intramuscular injections.
Bismuth salts	B	For oral or external use substitute aluminium hydroxide, chalk, kaolin and magnesium trisilicate.
Bismuthi Salicylas	B	Reserve for intramuscular injections.
Bism. Oxychlor.	B	Reserve for intramuscular injections.
Borax	B	In dermatological practice reserve for Unguentum Aquosum. Preparations of glycerin or honey may be replaced by watery solutions of aniline dyes in the treatment of thrush.
Brilliant Green	—	See Dyes.
Bromethol	A	Avertin is identical.
Bromides	A	
Bromoformum	C	
Buchu	C	
Caffeina	A	Omit from compound tablets such as compound aspirin tablets (A.P.C.).
Caffeina et Sodii Benzoas	C	Nikethamide (Coramine) and Leptazol (Cardiazol) are partial substitutes.
Calumba	C	Replace with alstonia, Cort. Aurant, lupulus, quassia.
Calamina	Aus.	
Calciferol	B	Use dietetic sources where possible.
Calcii Carbonas	Aus.	
Calcii Chloridum	Aus.	
Calcii Gluconas	B	Reserve for injections.
Calcii Glycerophos	C	Substitute Calcii Lactas.
Calcii Hydroxidum	Aus.	
Calcii Lactas	Aus.	
Calcii Sulphas Exsiccatus	Aus.	
Calx Chlorinata	Aus.	
Camphora	B	Reserve for Tr. Opii Camph. For external use substitute Ol. Eucalyptus.
Cannabis	C	
Cantharis	C	Substitute sinapis.
Cantharidinum	C	Substitute sinapis.
Capsicum	C	
Carbacholum	C	Doryl is identical.
Carbamide (urea)	A	
Carbo	Aus.	
Carbo Activatus	Aus.	
Carbonei Dioxidum	Aus.	
Carbonei Tetrachloridum	B	Reserve for use as an anthelmintic (hookworm).
Carbromalum	C	Substitute chloral and barbiturates.
Cardamomum	C	Substitute Tr. Aurant.
Cardiazol	—	Leptazolum and Metrazol are identical.
Caryophyllum	C	
Cascara Sagrada	A	
Cassia	C	
Catechu	C	Substitutes: kaolin, Calcii Carb.
Cera	Aus.	
Chiniofonum	A	Yatren is identical. (Alternative drugs for amœbic dysentery are emetine and acetarsol.)
Chloral Hydras	Aus.	
Chloramina	C	Substitutes: Dakin's solution or chlorocresol.
Chlorbutol	Aus.	
Chlorocresol (B.P.)	A	Parachlorometacresol is identical. Chlorinated xylenols are equivalent.
Chloroformum	A	
Chondrus	C	
Chromii Trioxidum (Acidum Chromicum)	C	
Chrysarobinum	A	
Cinchona	C	The use of cinchona as a bitter is unnecessary. Replace with alstonia.
Cinchophenum	C	Atophan and Agotan are identical.

Drug.	Classification.	Substitute or Equivalent and Remarks.
Cinnamomum	C	See Olea d. Essential oils.
Coca	C	
Cocaina and its salts	A	
Coccus	C	
Codeina and salts	A	
Colchici Cormus	A	Production should be encouraged in Australia.
Colchici Semen	A	
Colocynthis	C	Limited quantity of Australian grown available.
Colophonium	C	
Copaiba	C	Substitutes: sulphanilamide, hexamine, mandelic acid.
Coramine	—	Nikethamide is identical.
Coriandrum	C	Replace with Cort. Aurant, Cort. Limonis and Menth. Pip.
Corpus Luteum, Progesterone and preparations	B	Progesterone proprietary equivalents: Lipo-Lutin, Luteostab, Lutren, Progestin, Proluton.
Creosotum	A	
Cresol	Aus.	
Creta	C	Replace with Calcii Carb. Præcip. Kaolin is a substitute.
Cubeba	C	
Cupri Sulphas	Aus.	
Cyclopropanum	C	
Desoxycorticosterone and its esters	B	Proprietary equivalents: Cortenil, DOCA, Percortin.
Dextrosum B.P.	B	Reserve for intravenous injection.
Dial	—	Allobarbitonum is identical. See Barbiturates.
Diamorphinæ Hydrochlor.	A	
Digitalis	Aus.	Prescribe as standard powder (tablets) and tincture only.
Digoxinum	B	For percutaneous use only.
Dionin	—	Ethylmorphine is identical.
Doryl	—	Carbacholum is identical.
Dyes—		
Antiseptic	A	Brilliant green, crystal violet (gentian violet), flavines (acriflavine, euflavine and proflavine), fuchsine, malachite green, mercurochrome, methylthionine chloride, methyl violet and scarlet red.
Diagnostic	A	Fluorescein, indicarmine and methylthionine chloride.
Emetine	A	Acetarsol and chiniofon are partial equivalents.
Ephedra	—	Production should be encouraged.
Ephedrina and its salts	A	
Ergometrine	B	Reserve for percutaneous use.
Ergota	A	Standard powder (Ergota Preparata) in tablet form is the most stable preparation.
Euflavine	A	See Dyes, Antiseptic.
Evipan and Evipan Sodium	—	Hexobarbitonum and Hexobarbitonum Soluble are identical.
Extractum Fellis Bovini	Aus.	
Extractum Hepatis Liquidum (and other oral preparations of liver)	Aus.	See Liver extracts.
Extractum Malti	Aus.	
Ferri Carbonas	Aus.	Use to replace all other iron preparations.
Ferri Carbonas Saccharatus	Aus.	
Ferri et Ammonii Citras	Aus.	
Ferri Sulphas	Aus.	
Filix Mas and extracts	A	Production should be encouraged.
Fluoresceinum Soluble	A	See Dyes, Diagnostic.
Fouadin	—	Stibophen is identical.
Formaldehyde	B	Under Government control.
Galla	C	
Gelatinum	Aus.	
Gentian	C	Replace with Cort. Aurant and lupulus.
Germanin	—	Suramin is identical.
Gold Compounds for Parenteral use only	A	Auri et Sodii Thiosulphas, Sodium Aurothiomalate. Proprietary equivalents: Crisalbine, Myocrisin, Solganal, Sanocrysin.
Guaiacol	C	Substitute creosote.
Glycerinum	Aus.	Under Government control. For most purposes Acacia Tragacanth or starch may be substituted. In special cases use invert sugar or sodi. lactate.
Glycerylis Trinitras	A	Use tablets only.
Glycyrrhiza	Aus.	
Glucose Liquid	Aus.	For general use. Reserve dextrose B.P. for intravenous use.
Hamamelis	C	Substitute tannic acid.
Hexobarbitonum	A	Evipan is identical. See Barbiturates.
Hexobarbitonum Soluble	A	Evipan Sodium is identical.
Hexamina	A	
Hexyl Resorcinol	C	
Histamine Phosphas Acidus	C	
Homatropina and its salts	A	
Hydrargyrum	A	
Hydrargyrum Ammoniatum	A	
Hydrargyri Iodidum Rubrum	C	
Hydrargyri Oxidum Flavum	A	
Hydrargyri Oxycyanidum	C	
Hydrargyri Perchloridum	A	Substitute chlorocresol or chlorinated xylenols as antiseptic lotions.
Hydrargyri Subchloridum	A	
Hyoscyamus Niger	C	Substitute preparations of strammon. or belladon.
Hyoscina and its salts	Aus.	
Ichthammol	C	
Indicarminum	A	See Dyes, Diagnostic.
Insulinum	Aus.	
Insulinum Protaminatum cum Zinco.	Aus.	
Iodine compounds for X-ray diagnosis—		Proprietary equivalents for iodine compounds.
Iodophthalein	B	Opacin. Stipolac. T.I.P.
Iodoxylum	B	Uroselectan B, Tenebryl, Per-Abrodil.
Oleum Iodisatum	B	Iodatol, Lipiodol, Neohydriol, Skiagenol.
Iodoformum	C	Substitutes: proflavine, chlorocresol.
Iodoxylum	B	See Iodine Compounds.
Iodum	A	Reserve for internal use. Substitutes for external use, flavines and other antiseptic dyes.
Ipecacuanha	A	Should be reserved for the manufacture of emetine. Galenical preparations of ipecacuanha as expectorants and emetics are not essential. Substitute carbonates or chlorides of ammonium.
Jalapa	C	
Kaolinum	Aus.	
Kino	C	
Krameria	C	
Lactosum	Aus.	
Linum	Aus.	
Leptazolum	A	Cardiazol and phrenazol are identical.
Liquor Cresolis Saponatus (Lysol)	Aus.	
Liquor Ferri Perchloridi	Aus.	
Liquor Formaldehydi	B	Under Government control.
Liquor Glycerylis Trinitratis	C	Replace with tablets.
Liquor Hydrogenii Peroxidi	Aus.	
Liquor Picis Carbonis	A	
Liquor Vitamini A Concentratum		
Liquor Vitaminorum A et D	A	Ol. Hippoglossi in equivalent dose is a substitute.
Liquor Vitamini D Concentratum	B	For specific use in rickets.
Liver preparations—		
Dry and liquid extracts	Aus.	For oral use.
Liver extracts for parenteral use	B	Reserve for treatment of macrocytic anæmias. Proprietary equivalents are Anahæmin, Campolon, Examen, Hepatex, Reticulogen and others.
Lobelia	C	Substitute stramonium.
Luminal and Luminal Sodium	—	Phenobarbitone and soluble phenobarbitone are identical. See Barbiturates.
Lupulus (hops)	Aus.	Substitute for imported aromatic bitters.
Magnesii Carbonas	Aus.	
Magnesii Oxidum	Aus.	
Magnesii Sulphas	Aus.	
Magnesii Trisilicas	Aus.	
Medinal	—	Barbitone soluble is identical.
Mel	Aus.	
Menthol	A	
Mepacrinæ Hydrochloridum	B	See Quinine Salts.

Drug.	Classification.	Substitute or Equivalent and Remarks.
Mepacrinæ Methansulphonas	B	See Quinine Salts.
Mersalylum	A	Proprietaries: Salyrgan, Mercurgan are identical. Neptal, Merbaphen (Novasurol) are equivalent.
Methylis Salicylas	Aus.	
Methylsulphonal	C	
Methylthioninæ Chloridum	A	Methylene blue. See Dyes.
Morphina and its salts	A	
Myosalvarsan	—	See Arsenicals (organic). Sulpharsphenamine.
Nembutal	—	Pentobarbital Sodium is identical. See Barbiturates.
Neoarsphenamina	A	See Arsenicals (organic).
Nicotinamidum	B	Use dietetic sources where possible.
Nikethamide	A	Coramine is identical.
Nitrogenii Monoxidum	Aus.	
Novocaine	—	Procaine hydrochloride is identical.
Nux Vomica extracts	A	
Œstradiol, Œstriol, Œstrone and their esters	A	Proprietary equivalents: Œstroform, Œstroglandol, Progynon, Theelin, Ovocyclin.
Oleum Abietis	C	Substitute Ol. titree or huon pine.
Olea—		
a. Edible and emollient oils—		
Ol. Amygdalæ Australiensis	Aus.	All the edible and emollient oils are therapeutically equivalent and interchangeable.
Arachis	Aus.	
Gossypii	Aus.	
Maydis	Aus.	
Olivæ	Aus.	
b. Vitamin-containing oils—		Standardized to contain per gramme:
Hippoglossi	A	Vit. A, 30,000 units. Vit. D, 3,000 units.
Morrhuæ	A	Not less than: Vit. A, 600 units. Vit. D, 85 units.
Vitaminatum	A	Vit. A 1,000 units. Vit. D, 100 units. These in proportionate doses are equivalent in vitamins.
c. Other fixed oils—		
Ol. Lini	Aus.	
Ricini	Aus.	
Theobromatis	Aus.	Reserve for use in suppositories.
d. Essential oils—		
Ol. Anethi	A	
Anisi	A	
Cadinum	C	
Caryophylli	A	
Chenopodii	B	
Cinnamon	A	
Eucalypti	Aus.	
Caultheria	C	Methyl salicylate is identical.
Limonis	Aus.	
Menth. Pip	Aus.	
Santal Australiensis	Aus.	
Terebinth	C	For external use. Substitute Ol. Eucalypti.
Opium	A	
Orthocaina	C	Benzocaine is equivalent.
Ovarian hormones and preparations	A	See Œstradiol.
Oxygenium	Aus.	
Pamaquinum	B	See Quinine Salts.
Pancreatinum	A	
Paraffinum Liquidum	A	Its use should be restricted.
Paraffinum Molle	A	Substitute Ung. Simplex E.F.A.
Paraldehydum	A	
Parathyroidei	B	Proprietary equivalents: Paroidin, Parathormone.
Pentobarbital Sodium	A	See Barbiturates.
Pepsinum	C	
Per-Abrodil	—	Iodoxylum is identical. See Iodine Compounds for X-ray diagnosis.
Phenitone	A	Prominal is identical. See Barbiturates.
Phenacetinum	A	
Phenazonum	C	Substitutes: acetylsalicylic acid and phenacetin.
Phenobarbitonum	A	See Barbiturates.
Phenobarbitonum Soluble	A	See Barbiturates.
Phenol	Aus.	
Phenolphthaleinum	A	
Phenothiazine	A	
Phrenazol	—	Leptazol is identical.
Physostigmina and its salts	A	
Pilocarpina and its salts	A	
Pirevan	A	See Acaprin.
Pituitary posterior lobe extracts	Aus.	Ext. Pituitarii Liq. B.P.
Pix Carbonis Præparata	Aus.	Substitute Pix Carbonis.
Pix Liquida	C	
Plasmoquin	—	Pamaquinum is identical. See Quinine Salts.
Plumbi Acetas	A	
Podophylli Resina	C	
Potassa Sulphurata	Aus.	Substitutes: sulphur ointment, solution of sulphurated lime.
Potassii Bicarbonas	C	Substitute sodium bicarbonate.
Potassii Bromidum	A	Substitute sodium bromide.
Potassii Chloras	C	Not essential.
Potassii Citras	Aus.	Substitute sodium citrate.
Potassii Hydroxidum	C	Substitute sodium hydroxide.
Potassii Iodidum	A	Substitute sodium iodide.
Potassii Nitras	C	
Potassii Permanganas	B	Reserve for use as an oxidizing antiseptic.
Potassii Tartras Acidus	C	
Procainæ Hydrochloridum	A	Ethocaine, Neocaine, Novocain, Kerocaine, Planocaine are identical.
Proflavina	A	See Dyes, Antiseptic.
Prominal	—	Phemitone is identical. See Barbiturates.
Prontosil Album	—	Sulphanilamide is identical. See Sulphanilamides.
Protargol	—	Argent Proteinum is equivalent.
Progesterone	—	See Corpus Luteum.
Pyroxylinum	Aus.	
Quassia	C	Substitute Alstonia.
Quillaia	B	Reserve for Liq. Picis. Carb.
Quinidinæ Sulphas	A	
Quinine salts	A	Antimalarial equivalents: Mepacrine Hydrochloride (Atebrin), Pamaquin (Plasmoquin), Mepacrine Methanesulphonas.
Resorcinol	A	
Rheum	A	
Saccharinum Soluble	B	Reserve for diabetics.
Salicinum	C	Substitute salicylates.
Salol	C	Unnecessary.
Salyrgan	—	Mersalylum is identical.
Santoninum	A	
Scammonium	C	
Scilla	C	Substitute other diuretics or expectorants.
Scoparium	C	
Senega	C	Substitutes: ammonium carbonate, ammonium bicarbonate, ammonium chloride.
Senna leaves and pods	A	
Sera, vaccines and biological diagnostic reagents	Aus.	Commonwealth Serum Laboratories provide all essential requirements.
Sinapis	A	
Sodii Aminarsonas	C	(Atoxyl). See Arsenicals (organic).
Sodii Benzoas	C	Substitute mandelates or sulphanilamides.
Sodii Bicarbonas	Aus.	
Sodii Bromidum	A	
Sodii Carbonas	Aus.	
Sodii Chloridum	Aus.	
Sodii Citras	Aus.	
Sodii Iodidum	A	
Sodii Lactas	Aus.	Use as glycerin substitute in cataplasma kaolin, etc.
Sodii Nitris	C	Substitute glyceryl trinitrate tablets.
Sodii Phosphas	C	
Sodii Phosphas Acidus	A	
Sodii et Potassii Tartras	A	
Sodii Salicylas	A	
Sodii Sulphas	Aus.	
Sodii Tauroglycocholas	C	Substitute Felis Bovini (extract of ox bile).
Sodii Thiosulphas	A	
Sparteina	C	
Spiritus Ætheris Nitrosi	Aus.	
Stannum	C	
Stanni Oxidum	C	
Stibophenum	B	Proprietary equivalent, Fouadin.
Stilbœstrol	A	
Stramonium	A	
Strophanthus	C	Substitute digitalis.
Strychnina and its salts	A	
Styrax	B	Reserve for compound tincture of benzoin.
Sucrosum	Aus.	

Drug.	Classification.	Substitute or Equivalent and Remarks.
Sulphanilamides—Sulphanilamide	A	Proprietary equivalents: Prontosil Album, Streptocide, Sulphonamide P.
Sulphaguanidine	A	
Sulphapyridine	A	Dagenan, M & B 693.
Sulphathiazol	A	Thiazamide, M & B 760.
		Unclassified: Albucid (sulphacetamide), Prontosil Rubrum, Prontosil Soluble, Proseptasine, Soluseptasine, Uleron.
Sulpharsphenamina	A	See Arsenicals (organic).
Sulphonal	C	Substitutes: barbiturates, chloral hydrate, chlorbutol.
Sulphur	A	
Suprarenal cortex and preparations	B	See Desoxycorticosterone.
Suraminum	A	Proprietary equivalents: Bayer 205, Germanin.
Syrupus Ferri Phosphatis Composita	B	Reserve for children.
Syrupus Ferri Phosphatis cum Quinina et Strychnina	C	
Talcum	Aus.	
Taraxacum	C	
Theobromina	A	
Theobromina et Sodii Salicylas	A	Proprietary equivalent: Diuretin.
Theophyllina et Sodii Acetas	A	Theocin sodium acetate is identical, Theophyllina cum Æthylene Diamina is equivalent. Proprietary equivalents: Euphyllin, Metaphyllin.
Theophyllina	A	
Thymol	A	
Thyroideum	Aus.	
Thyroxinsodium	C	
Tragacantha	B	Reserve for medicinal preparations.
Trinitrophenol	A	
Tryparsamidum	A	See Arsenicals (organic).
Urea	A	
Uroselectan B	—	See Iodine Compounds for X-ray diagnosis.
Valeriana	C	
Veronal	—	Barbitonum is identical. See Barbiturates.
Ventriculus Disiccatus	Aus.	
Vitamins	—	See Acidum Ascorbicum, Aneurinæ, Hydrochloridum, Calciferol, Liquor Vitamini A Concentratum, Liquor Vitamini D Concentratum, Liquor Vitaminorum A et D Concentratum, Acidum Nicotinicum, Oleum Hippoglossi, Oleum Morrhuæ, Oleum Vitaminatum.
Yatren	—	Chiniofon is identical.
Zinci Carbonas	C	Calamine is equivalent.
Zinci Chloridum	Aus.	
Zinci Oxidum	Aus.	
Zinci Stearas	Aus.	
Zinci Sulphas	Aus.	
Zingiber	C	

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 15, of January 15, 1942.

AUSTRALIAN MILITARY FORCES.

AUSTRALIAN ARMY MEDICAL CORPS.

Northern Command.

First Military District.

The resignation of Honorary Captain P. A. Foley of his commission is accepted, 6th December, 1941.

Eastern Command.

Second Military District.

Honorary Captain N274189 A. M. Bryson is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 2nd December, 1941.

The following officers are retired: Major A. J. Collins, D.S.O., M.C., Captain (Honorary Lieutenant-Colonel) G. Bell, O.B.E., 30th November, 1941; and Captain R. D. Mulvey, M.C., 31st December, 1941.

To be Honorary Majors.—Honorary Captains F. G. Roberts and B. P. Anderson-Stuart, 15th October, 1941.

To be Honorary Captains.—Edwin Steuart Welch, William Matthew James Quinn, 1st December, 1941; Douglas Godfrey Watson, 8th December, 1941; and John Michael Garvan, 15th December, 1941.

Southern Command.

Third Military District.

Honorary Captain C. L. Coghlan is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 1st November, 1941.

The resignation of Honorary Captain W. A. McKay of his commission is accepted, 1st December, 1941.

Fourth Military District.

Honorary Captain S997 E. J. K. Harbison is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 30th July, 1940.

Honorary Captain W. C. Sangster is appointed from the Reserve of Officers (A.A.M.C.), to be Captain (provisionally), 24th September, 1941, and to be Major (temporarily), 9th December, 1941.

To be Major (temporarily).—Captain (provisionally) A. J. Blofeld-Moody, 10th December, 1941.

Sixth Military District.

Captain J. L. Grove is appointed from the Reserve of Officers (A.A.M.C.), and to be Major (temporarily), 9th December, 1941.

The resignation of Honorary Captain J. M. Gaskell of his commission is accepted, 28th August, 1941.

DECORATIONS.

LIEUTENANT-COLONEL John George Glyn White, of Victoria, has been created an Officer of the Most Excellent Order of the British Empire.

The Director of Naval Medical Services announces that Surgeon Lieutenant-Commander Eric Mortimer Tymms, of the Royal Australian Naval Reserve, was awarded the Distinguished Service Cross in the list of awards published in the New Year. The late Surgeon Lieutenant Charles Frederick Harrington was awarded the Distinguished Service Cross at the same time. Surgeon-Lieutenant Harrington was killed in action in H.M.A.S. *Parramatta*.

Obituary.

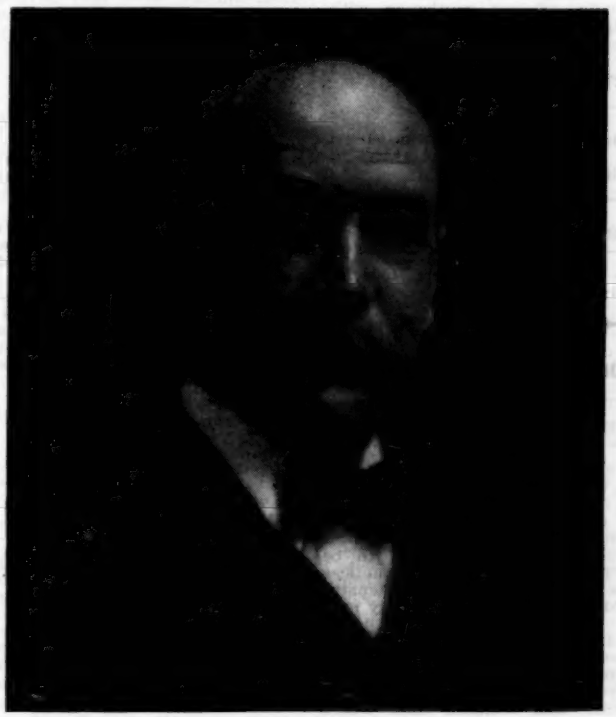
WILLIAM CHISHOLM.

SYDNEY graduates in medicine of the early years of this century remember William Chisholm as a surgeon who was gifted with patience and skill, who was "safe" in an emergency, and who worked in ward and operating theatre with slow deliberation and with almost a courtly grace. His whole life seemed to be lived in this way; he belonged to what the older practitioners of today choose to call the old school—he was neither loud of voice nor violent of manner, he had no ostentation, he came to do his work, he did it without fuss and went quietly, almost slipped away, when he had finished. He was like this when in due course he gave up hospital practice; his help was there when it was wanted, and he gave, as he always had given, freely and with an open hand. In the evening of life he slipped away from the public gaze, and among those whom he loved was able, in words that he once quoted in an address to students, to

> . . . . approach the grave
> Like one who wraps the drapery of his couch
> Around him, and lies down to pleasant dreams.

William Chisholm was born not far from Wallergerang, near Goulburn, New South Wales, in 1853. He was the

son of the late John William Chisholm, grazier. He went to the King's School when it was situated at Macquarie Fields and James MacArthur was the headmaster. Later the school was moved to Parramatta. In 1870-1871 William Chisholm was captain of the school. In 1872 he passed on to Saint Paul's College and studied Arts at the University of Sydney, where he graduated as Bachelor of Arts three years later. He went to England to study medicine and was admitted as Member of the Royal College of Surgeons of England in 1880. He graduated as Doctor of Medicine in 1883. Having returned to Sydney, he joined the surgical staff of Saint Vincent's Hospital. He served this institution for some years and retired with some other members of the staff as the result of a disagreement. He served three other hospitals in Sydney. In 1889 he became honorary surgeon to the Sydney Hospital for Sick Children. This institution is now known as the Royal Alexandra Hospital for Children; in those days it was situated in Glebe Road. In his work at the Sydney Hospital for Sick Children Chisholm was associated with the late Charles Percy Barlee Clubbe. Chisholm in many ways resembled Clubbe. He had the same kindliness of manner, and children loved him as they did Clubbe. Of the two men Chisholm was the more retiring in disposition and the more deliberate in his work—he sometimes seemed to be over-careful, fearful lest he should make a mistake—but be that as it may, his name is remembered today with honour and affection, as are those of Clubbe and Tidswell. From 1889 to 1894 Chisholm served on the staff of Prince Alfred Hospital (it had not then been dignified by the title "Royal") and in 1894 became honorary surgeon at Sydney Hospital. He was associated with this institution until his death. As honorary surgeon from 1894 to 1911 he set an example of sound surgery; he taught his resident medical officers more by example than by precept, and those whose minds were receptive gained more than they would have gained from one who might have "dressed his window" better or have shown what is commonly termed brilliance. In 1911 he became honorary consulting surgeon and retained the position till his death. From 1903 to 1908 he was a member of the Board of Directors of Sydney Hospital, and in 1930 the William Chisholm Operating Theatre in the Travers Pavilion was named after him.



In 1910 William Chisholm went to England. He was studying in Vienna in 1914 just before the outbreak of war. He at once offered his services to the Royal Army Medical Corps; later on he transferred to the Australian Army Medical Corps. He served on the staff of the Australian Voluntary Hospital at Wimereux under Colonel W. L'Estrange Eames, and later on did useful work at the Australian Stationary Hospital at Southall, London. His son Lieutenant W. W. Chisholm was killed at Mons on August 26, 1914—the first Australian to be killed in France during the Great War—and this was a blow which told very heavily on him and from which he never quite recovered.

The foregoing has shown what manner of man William Chisholm was. One of his old resident medical officers has said that William Chisholm, almost more than any of the surgeons of his day, showed forth in his practice the mind of a physician. This is probably true. It is quite certain that Chisholm had trained his powers of observation and that his clinical sense was highly developed. In the address to students already mentioned he said:

> Cultivate your faculty of observation. There is more to be learnt from the manner of respiration than the mere number of inspirations in a minute; so too the quality of a pulse will often tell you more than its frequency and you can learn much from the expression of a countenance.

These were words which Chisholm put into practice. Sometimes he was slow to make a diagnosis, but, as Dr. Edwards writes in his personal appreciation, once the opinion was given he would stick to it. He had human sympathy and frequently would visit the anæsthetic room in order to give courage to a patient who was fearful. To his students he said:

> It will be well always to bear in mind that a hospital ward is a place of suffering where you may learn to admire the patience with which much of it is borne and, though many a patient may have what seems to you a trivial complaint, remember that it is not the disease but a being of like parts and passions as yourself that you are treating, and you may not know the deadening influence of anxiety on some breadwinner laid temporarily aside by illness.

Like a true clinician, Chisholm always put the patient first. His early days in surgical practice were those of the pre-Listerian period, when, as he himself has related, a surgeon who inadvertently opened the peritoneal cavity in the course of an operation, thought he had killed his patient. Some who were introduced to surgery before the days of Lister found it difficult to adopt the new practices and were slow to bring to their aid other discoveries as they were made. Chisholm had an open mind, he followed what was new and proven good, but was conservative enough to use old methods when he knew they would be useful. When he said: "Upon my soul, do you know, I think we'll order him a linseed meal poultice", the prescription generally proved effective. He disliked pretence and cared not for appearances of erudition unbacked by experience. He wrote:

> "Do not be anxious to appear 'up to date' by too great readiness in adopting the latest scientific methods. A wise old doctor once said: 'Science is a first-rate piece of furniture for a man's upper chamber, if he has common sense on the ground floor. But if a man has not got plenty of good common sense, the more science he has, the worse for his patient.'"

William Chisholm has passed from us. Many of the present-day practitioners did not know him. Not to have known and worked for men of his calibre is a deficiency which nothing can make good. He and others like him have set a high standard of character and achievement; to attain such a standard is a worthy goal. Chisholm himself said: "In most things if you aim at superiority you will be sure to rise above mediocrity; if you are content to aim only at mediocrity, rest assured you will fall below it." He would have fled from the idea that anything he had done was of superior worth, but the fact remains that his life gave to others "matter for virtuous deeds".

Dr. J. G. Edwards writes:

It was my privilege to be closely associated with William Chisholm from 1907 to 1913 in his professional work as senior honorary surgeon to the Sydney Hospital.

No man ever gave better service to his patients, and at any time of the day or night he might be called upon for urgent work and would respond without a single murmur of protest. Of all the surgeons I have met, I am certain that none ever surpassed him in soundness of opinion in abdominal emergencies. To me he always appeared to possess "second sight" in this branch of surgery. A rigid

abdominal wall held no secrets for him. If Chisholm decided to "leave" an urgent abdominal case, nobody worried any more; but if he decided to operate, another life was saved.

His sound opinion was always delivered in a hesitant manner, but once it was given it was impossible to shift him; often we tiros with the enthusiasm of youth would argue *as contraire*, but to no avail. I once asked him why he decided to leave a clear case of ruptured viscus, and his reply was: "Upon my soul, if I operate he will die, but if I leave him he may recover"; he recovered.

As a surgical technician he was slow but extraordinarily neat—he was an accomplished "seamstress", and it was notorious how little shock his patients suffered. He was as gentle in manipulation as he was in manner. During his surgical life he kept well in step with advancing thought and technique. To house surgeon and patient he was invariably courteous, and those of us who worked with him will never forget his kindly manner. If ever the term "Nature's gentleman" was applicable it was so in the case of William Chisholm, and it is a great privilege to have been associated with this kindly, efficient gentleman with his profound surgical knowledge.

JOHN STUART CAMPBELL.

WE regret to announce the death of Dr. John Stuart Campbell, which occurred on January 20, 1942, at Marrickville, New South Wales.

ROBERT RODERICK HARDMAN.

WE regret to announce the death of Dr. Robert Roderick Hardman, which occurred on January 24, 1942, at Sydney, New South Wales.

Correspondence.

A SALARIED MEDICAL SERVICE.

SIR: The National Health and Research Council is to be congratulated on the facility with which they have handled figures and statistics in their communication to THE MEDICAL JOURNAL OF AUSTRALIA under the heading of Public Health, "An Outline of a Possible Scheme for a Salaried Medical Service", appearing on December 20, 1941. It is to be regretted that their statistics regarding local practices suggest either that their researches are not very extensive or that caustic remarks regarding statisticians generally may be justified.

I can only speak with definite knowledge of the following practices: Wongan Hills, Goomalling, Dowerin, Koorda and Dalwallinu. Of these five towns, all of which have had hospitals for more than six years, Dowerin and Koorda are classed as not having hospitals; and Wongan Hills, where I have been in practice since early 1936, is stated to have no doctor. The true facts could easily have been obtained from the local branch of the British Medical Association or from the Department of Public Health.

Small slips like these suggest that more care could have been exercised before rushing into print; and it is to be hoped that other statistics are more accurate.

The suggestion that much domiciliary visiting may be necessary in A class centres may be true as far as old-established districts are concerned; but in Western Australian wheat-belt practices it is usually advisable to admit sick people to hospital; the houses are often shacks, unbearably hot in summer and draughty in winter; and domestic assistance is unobtainable in the event of the wife being sick.

It will be necessary to train a far larger number of nurses than hitherto (on the supposition that two extra will be required for each B class centre this means at least an extra seventy in Western Australia for these centres alone); and it has always been difficult to obtain nursing staffs for country hospitals.

I would like to protest against the suggestion that administrative chiefs should receive salaries in any way comparable with consultant specialists. The consultant specialist not only has to possess exceptional clinical ability, but usually has to pass examinations of the standard of the English F.R.C.S. or M.R.C.P. Having attained this skill, he should not be tempted to desert the work he does best for work that many able doctors do badly—administration. The administrative chief, I presume, would be content with a D.P.H. and sufficient business training to put into effect the useful discoveries of the more gifted members of our profession (the clinicians, pathologists and bacteriologists who do the original work). Personally I believe that clinical ability and the ability to do original research work should be rewarded more liberally than business ability; but this is of course a personal opinion.

If a salaried service is provided, presumably private practitioners will be forced to join owing to the rather unfair competition—the people who formerly belonged to his practice having had to pay taxes will be eligible for free treatment and are unlikely to be able to afford much in the way of "extras", for example, free choice of doctor. Some method of purchasing the practices of men thus forced out of private practice should be considered.

It would, in any case, be wise to wait till our professional brethren in the armed forces return to practice. They have had personal experience of a salaried medical service and their opinion, after they have been discharged from service (not earlier) will be of great value.

Yours, etc.,

T. LOVEGROVE.

Wongan Hills,
Western Australia,
January 3, 1942.

SIR: To me the important thing about any new scheme for medical practice is that it should be under the control of the profession itself and should not introduce outside domination.

Surely it is not asking too much for the Association to propound a concrete scheme which could be introduced at first on a voluntary basis and which could be used as a basis for later proposals by the Government. Such a procedure would firstly provide a definite basis for negotiation over the financial aspects and prevent a repetition of the 11s. a year fiasco, and secondly, the Association by providing the initiative should be able to retain at least a controlling interest in the scheme adopted.

Yours, etc.,

D'ARCY N. SHORT.

Adelaide Street,
Blayney,
New South Wales.
January 10, 1942.

HERPES RECURRENS.

SIR: In reply to Dr. Guy Griffiths's questions about *herpes recurrens* (THE MEDICAL JOURNAL OF AUSTRALIA, December 27, 1941), all recurrent herpes that have been investigated have provided virus of the *herpes simplex* type. There are a few instances on record where implantation of virus in some new region of skin, either accidentally or with therapeutic aim, has been followed by the development of recurrent herpes at the site of implantation.

The characteristics of the *herpes simplex* virus seem to make it futile to attempt any type of immunization or chemotherapeutic treatment. The indefinite persistence of the virus in the body has to be accepted, and therapeutic action can only be directed toward reducing the number of symptomatic attacks. There are such a multitude of possible causes which may wake the virus into activity that the only line of approach is to determine if possible what provokes the attacks in the individual patient and advise accordingly.

Yours, etc.,

F. M. BURNET.

The Walter and Eliza Hall Institute of Research
in Pathology and Medicine,
Royal Melbourne Hospital,
Melbourne.
January 12, 1942.

ATTEMPTED SUICIDE.

SIR: While being greatly appreciative of the collection of valuable statistical information about those who have attempted suicide, presented by Dr. J. G. McGeorge in his article (THE MEDICAL JOURNAL OF AUSTRALIA, January 17), I believe that the conclusions expressed call for some comment.

1. The article contains a great many pertinent facts concerning the problem of attempted suicide, but the questions of causation receive only casual mention. To say that hysteria, escape from boredom, depression, are reasons for suicide is only a superficial presentation, and ignores the analysis of the real motives, which are unconscious and therefore not known to the patient, although they may be readily discoverable by the psychiatrist. As Dr. McGeorge indicates, many cases are psychoneurotic, therefore their abnormal act must then be complex—determined; as he says, they may be legally sane, but to suggest that they are of sound mind is to adopt a cynical and reactionary viewpoint.

2. Dr. McGeorge's remarks on prevention betray the same cynical outlook. Of course, we cannot stop suicide; neither can we substantially reduce its incidence without reducing the incidence of psychoneurosis, pubertal maladjustments, and schizophrenia. Most psychiatrists believe that this reduction is possible and could be brought about by a change in certain of our social attitudes, the nature of which change implies a reorganization of economic, political and educational values. One does not need to be a master of hyperbole to concur with the main body of medical psychological knowledge to this extent; neither must one be a Utopian to hope that such change is imminent.

Yours, etc.,
DONALD BUCKLE.

Mental Hospital,
Stockton,
New South Wales.
January 17, 1942.

A NATIONAL MEDICAL SERVICE.

SIR: Dr. Paul Dane is deeply grieved at the import of an isolated sentence taken from my letter in THE MEDICAL JOURNAL OF AUSTRALIA of December 13, 1941: "If health cannot be 'sold' to the community it must be forced on it." By mischance this sentence is placed at the beginning of a paragraph, whereas it was intended to end the preceding paragraph, to the context of which it applies. In justice to myself and to avoid a possible error by Dr. Dane, may I ask for space to quote the full paragraph.

"If we study the present State control of public health we find that it is mostly cut to the measure of the voluntary response of the community; hence progress is slow and often wilfully obstructed. The approach by appeal to the community for its voluntary effort and support admits an individualism which is incompatible with the greater and more extensive welfare to be enjoyed under collectivism. The voluntary system presumes a standard of intelligence which does not exist, and permits the individual to judge of the value of certain highly scientific measures, the product of long research, which might be undertaken for the common good. How often we find that the individual's judgement is not guided by special knowledge or acceptance of the lead of others in scientific thought, but by exasperating prejudice, ignorance or false religious scruples. Those who still believe in astrology and fondle mystic charms can be hardly expected to grasp the significance of mass immunization against devastating epidemics. If health cannot be 'sold' to the community it must be forced on it."

I suppose the use of such words as individualism and collectivism may startle many a gentle reader, but is anything more painful to witness in our days of peril than the capricious individualism underlying the strikes of today, and is anything more admirable than the collectivism of our fighting services? It is wrong to accuse me of being an advocate of socialization; the existing order suits me well; few have enjoyed more benefits and comforts under it, but I see changes ahead which will be out of our control. The correspondence I have conducted may have been provocative, but its one purpose has been to appeal to the members of the medical profession to take up a forward and strong strategic position in preparation for coming changes and not be dumb victims of a rapid political move. I stress the importance of studying political thought; we must sit in the councils which will determine our future so that our voices will be heard in discussion and deliberation, not in lamentation at the hard lot that has been thrust on us.

Yours, etc.,
JOHN MAUDE.

British Medical Association House,
135, Macquarie Street,
Sydney.
January 19, 1942.

THE WASTE OF ANTISEPTICS.

SIR: Under the heading "National Emergency Measures" in THE MEDICAL JOURNAL OF AUSTRALIA, January 17, 1942, page 93, a request is made for the prevention of the waste of antiseptics in operating theatres. Those who are not aware of the fact may be interested to learn that in the pre-operative preparation of the unbroken skin there is no need to use any other agent than soap, applied either with a brush, as in scrubbing the hands, or, where the anatomy of the part requires it, with a rough cloth.

The efficacy of this method has been proven by the writer in public and private work during the last eighteen months.

Yours, etc.,
J. L. ROBERTS.

Conway Street,
Lismore,
New South Wales.
January 19, 1942.

A.R.P. PRECAUTIONS.

SIR: I would like to urge that in all cases where large hospitals are expected to deal with casualties, some provision be made for at least one surgical honorary to sleep on the premises and that surgeons attached to such hospitals be forbidden to travel by car during air raids. My reasons for suggesting this interference with the surgeons' liberties are as follows:

1. The honorary is on the spot in case of emergency, and medical superintendents and other interested parties are not left speculating as to whether the surgeon will arrive.

2. There is already a serious depletion of medical man power and it is safer for the surgeon to be at his post instead of being exposed to the added unnecessary risk of the road in blackouts. I quote from THE MEDICAL JOURNAL OF AUSTRALIA, January 10, 1942, page 60, in which Dr. P. D. Wilson described to Dr. Hamilton the type of injuries dealt with at the Park Prewett Hospital, "a great many injuries and many fractures, the latter chiefly arising from accidents sustained while driving during the blackout".

3. The surgeon on the road during air raids can only be a nuisance to air-raid wardens and a menace to other road users who are necessarily on the road in the course of their duties, for example, firemen, ambulances *et cetera*.

I have not suggested that all honoraries sleep at their post of duty because I feel that a fair proportion of serious casualties will do better if given a chance to get over their initial shock before operation; but a certain number will need immediate operation and the others will need to be seen.

In my original suggestion at a local meeting of the British Medical Association I suggested that the honorary be present at hospitals from dusk till dawn, but was told this was impracticable—no reason given. I have therefore amended the suggestion to "sleep on the premises", as this will involve no hardship apart from some lack of home comforts.

My suggestion, of course, only applies to hospitals where honoraries do not live within walking distance of the hospital; it is not original or new, as it has been tried out in Liverpool and found valuable at least twelve months ago.

I trust that surgeons in other States will show more consideration than ours for the welfare of the community, as at present, at any rate, a live specialist is more valuable than a dead or injured hero.

Yours, etc.,
T. LOVEGROVE.

Wongan Hills,
Western Australia,
January 19, 1942.

MAN POWER.

SIR: One can only find excuse in the remarks of Sir James Barrett—*re* the fitness of men for military service—in the fact that possibly time has dimmed his memory, if such is not the case, one wonders what his policy has cost in repatriation in the way of pensions, hospitalization and the cost of trained staff to care for such people before they actually do die.

Any medical man that sends a soldier into the line that has any disability, from which he will die in six months, must know that before death there is usually a period—and probably a considerable period of that six months—when he will first of all hamper either an advancing or retreating force and after this a further period when he will take up valuable hospital space and the time of trained personnel which could be better employed attending men that could be made fit.

I am sure that 99% of medical men will agree that such a policy as advocated by Sir James Barrett would lead to complaints, and rightly so, as it could only act as a brake to the efficient functioning of any trained body of men.

In expressing these views I do so in a dual capacity, namely, as an officer in an infantry regiment and Royal Flying Corps in the last war, and as an examining medical officer in this.

Yours, etc.,

G. MAURICE DAVIS.

Ruthergien,
Victoria,
January 25, 1942.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Friedman, Icyk Mayer (registered January 7, 1942), 23, Adelaide Street, Bellevue Hill.

Begg, John Henry, M.B., 1941 (Univ. Sydney), The Prince Henry Hospital, Little Bay.

Books Received.

"Diseases of the Nose, Throat and Ear: A Handbook for Students and Practitioners", by I. S. Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E.; Second Edition; 1941. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 462, with 74 illustrations and 4 coloured plates. Price: 15s. net.

"A Short Textbook of Surgery", by C. F. W. Illingworth, M.D., Ch.M., F.R.C.S.Ed.; Third Edition; 1942. London: J. and A. Churchill Limited. Royal 8vo, pp. 702, with 12 special plates and 201 illustrations. Price: 27s. net.

"Anatomy and Physiology for Nurses", by W. G. Sears, M.D., M.R.C.P.; 1941. London: Edward Arnold and Company. Crown 8vo, pp. 383, with illustrations. Price: 6s. net.

"Treatment of the Patient Past Fifty", by E. P. Boas, M.D.; 1941. Chicago: The Year Book Publishers, Incorporated. Demy 8vo, pp. 324, with 19 special plates. Price: $4.00 net.

"My Love Must Wait: The Story of Matthew Flinders", by E. Hill; 1941. Sydney: Angus and Robertson Limited. Demy 8vo, pp. 463. Price: 8s. 6d. net.

"The Timeless Land", by Eleanor Dark; 1941. London: Collins; Sydney: Angus and Robertson Limited. Demy 8vo, pp. 449. Price: 10s. 6d. net.

"Furred Animals of Australia", by E. Troughton, F.R.Z.S., C.M.Z.S.; with 25 plates in colour by N. W. Cayley, F.R.Z.S.; 1941. Sydney: Angus and Robertson Limited. Royal 8vo, pp. 401. Price: 14s. 6d. net.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Adderton, Dorothy Isabel Orion, M.B., B.S., 1941 (Univ. Sydney), Hornsby District Hospital, Hornsby.

Aiken, James Edwin, M.B., 1941 (Univ. Sydney), Western Suburbs Hospital, Croydon.

Ariotti, Louis Charles Anthony, M.B., 1941 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Bailey, Alan William Hale, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.

Bartlett, Francis Kelvin, M.B., B.S., 1941 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Beaumont-Haynes, John, M.B., B.S., 1941 (Univ. Sydney), Western Suburbs Hospital, Croydon.

Begg, John Henry, M.B., B.S., 1941 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Bell, Malcolm, M.B., B.S., 1941 (Univ. Sydney), 4, Gipps Street, Wollongong.

Benson, Lois Ellen, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Sydney.

Black, Michael James Morrison, M.B., 1941 (Univ. Sydney), 12, Sunderland Flats, 157, Victoria Road, Bellevue Hill.

Blacket, Ralph Beattie, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Diary for the Month.

FEB. 4.—Western Australian Branch, B.M.A.: Council.
FEB. 5.—South Australian Branch, B.M.A.: Council.
FEB. 10.—Tasmanian Branch, B.M.A.: Branch.
FEB. 13.—Queensland Branch, B.M.A.: Council.
FEB. 18.—Western Australian Branch, B.M.A.: Branch.
FEB. 26.—South Australian Branch, B.M.A.: Branch.
FEB. 27.—Queensland Branch, B.M.A.: Council.
FEB. 27.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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